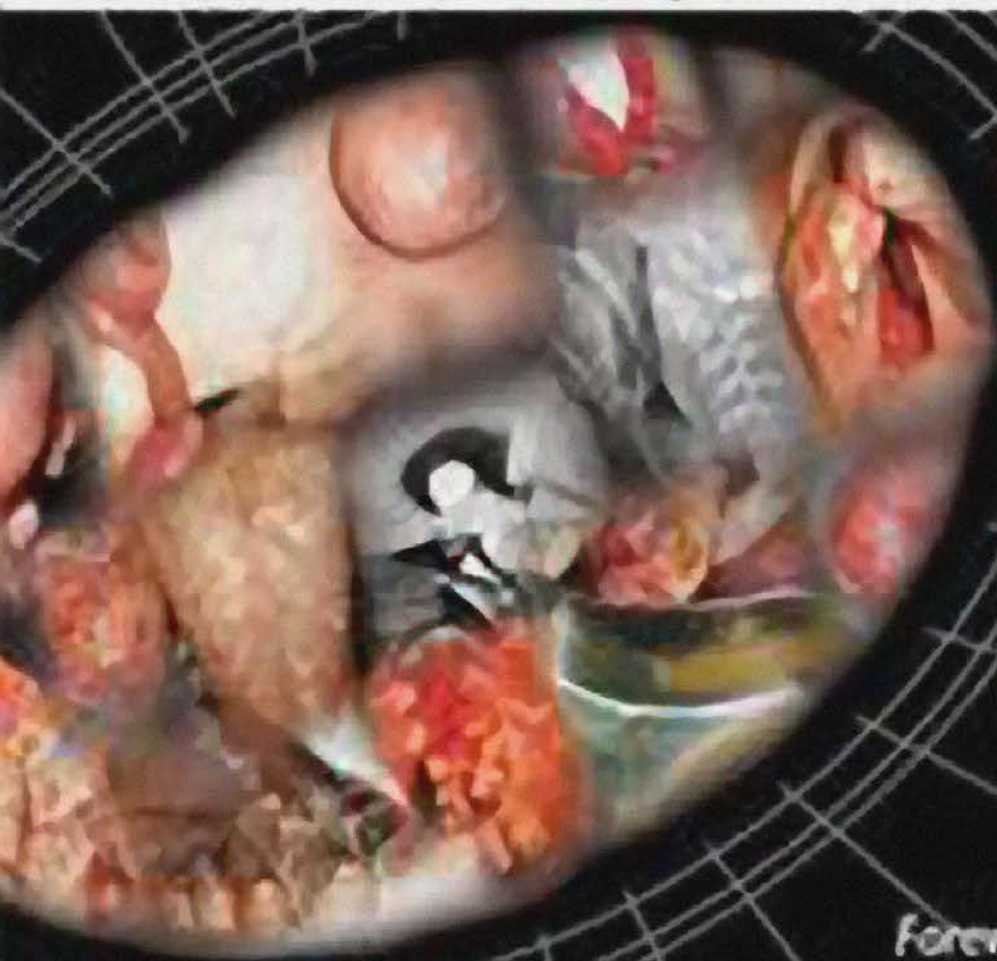


SRB's CLINICAL METHODS IN SURGERY

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Foreword CR Ballal

Sriram Bhat M

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SRB's
Clinical Methods in
SURGERY

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Clinical Methods in

SURGERY

Sriram Bhat M MS (General Surgery)
Additional Professor, Department of Surgery
Kasturba Medical College, Mangalore (Karnataka)
and
Honorary Surgeon
Government District Wenlock Hospital
Mangalore, Dakshina Kannada, India



JAYPEE BROTHERS MEDICAL PUBLISHERS (P) LTD

Bengaluru • St Louis (USA) • Panama City (Panama) • UK • New Delhi • Ahmedabad
Chennai • Hyderabad • Kochi • Kolkata • Lucknow • Mumbai • Nagpur

Published by

Jitendar P Vij

Jaypee Brothers Medical Publishers (P) Ltd

Corporate Office

4838/24 Ansari Road, Daryaganj, **New Delhi** - 110002, India

Phone: +91-11-43574357, Fax: +91-11-43574314

Registered Office

B-3 EMCA House, 23/23B Ansari Road, Daryaganj, **New Delhi** - 110 002, India

Phones: +91-11-23272143, +91-11-23272703, +91-11-23282021

+91-11-23245672, Rel: +91-11-32558559, Fax: +91-11-23276490, +91-11-23245683

e-mail: jaypee@jaypeebrothers.com, Website: www.jaypeebrothers.com

Offices in India

- **Ahmedabad**, Phone: Rel: +91-79-32988717, e-mail: ahmedabad@jaypeebrothers.com
- **Bengaluru**, Phone: Rel: +91-80-32714073, e-mail: bangalore@jaypeebrothers.com
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- **Hyderabad**, Phone: Rel: +91-40-32940929, e-mail: hyderabad@jaypeebrothers.com
- **Kochi**, Phone: +91-484-2395740, e-mail: kochi@jaypeebrothers.com
- **Kolkata**, Phone: +91-33-22276415, e-mail: kolkata@jaypeebrothers.com
- **Lucknow**, Phone: +91-522-3040554, e-mail: lucknow@jaypeebrothers.com
- **Mumbai**, Phone: Rel: +91-22-32926896, e-mail: mumbai@jaypeebrothers.com
- **Nagpur**, Phone: Rel: +91-712-3245220, e-mail: nagpur@jaypeebrothers.com

Overseas Offices

- **North America Office, USA**, Ph: 001-636-6279734, e-mail: jaypee@jaypeebrothers.com
anjulav@jaypeebrothers.com
- **Central America Office, Panama City, Panama**, Ph: 001-507-317-0160
e-mail: cservice@jphmedical.com, Website: www.jphmedical.com
- **Europe Office, UK**, Ph: +44 (0) 2031708910
e-mail: dholman@jpmedical.biz

SRB's Clinical Methods in Surgery

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First Edition: 2010

ISBN 978-81-8448-925-5

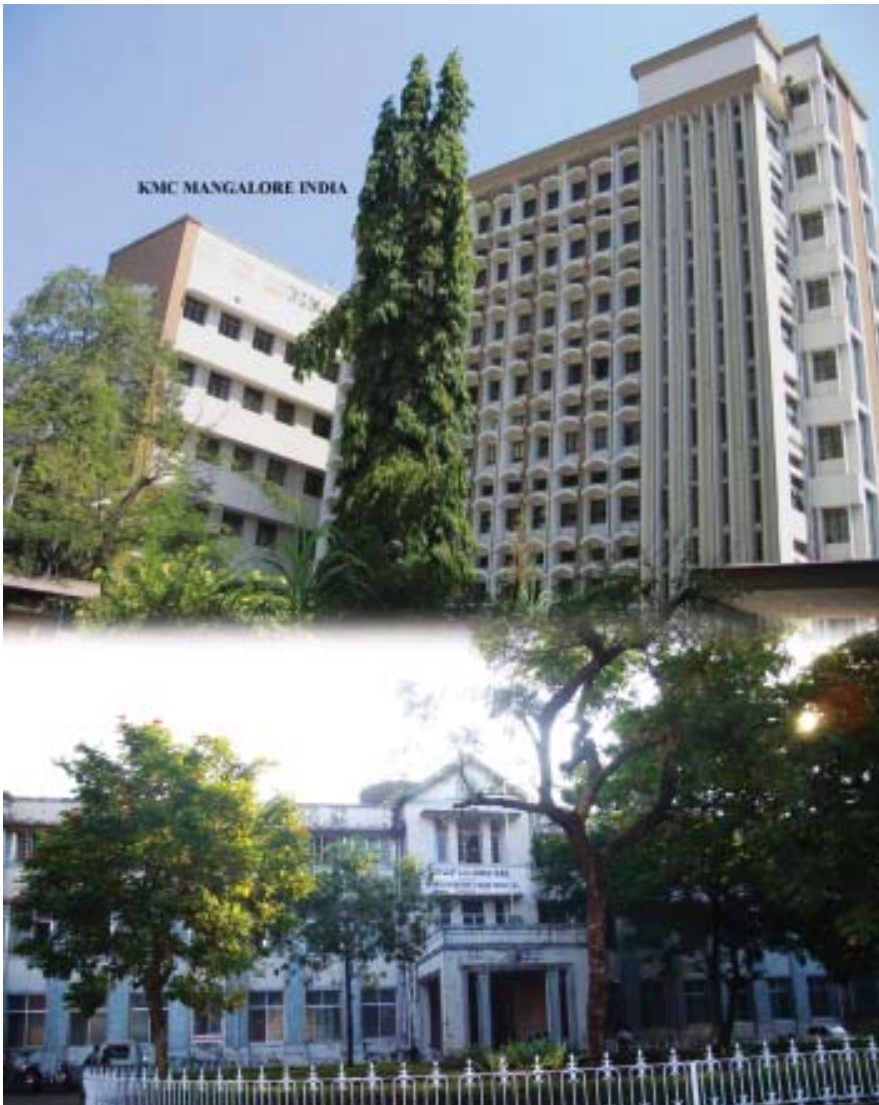
Typeset at JPBMP typesetting unit

Printed at

Dedicated to

College where I had my undergraduation, postgraduation and surgical professional career till date, i.e.

**KASTURBA MEDICAL COLLEGE
AND
DISTRICT WENLOCK HOSPITAL
MANGALORE, KARNATAKA, INDIA**



Foreword

Dr Sriram Bhat, Additional Professor of Surgery, KMC, Mangalore is a very popular teacher and I have known him for more than three decades. He has always been interested in teaching and his emphasis was on the fundamentals of good clinical examination. At a time when imaging and other methods of investigations are becoming popular than methodical and a well-executed clinical examination, a well-written book *SRB's Clinical Methods in Surgery* is most welcome. Dr Bhat has tried to emphasize a very systemic examination so that one could arrive at a working diagnosis at the end of a good history and physical examination. The value of this book has been enhanced by a very large number of highly illustrative pictorial descriptions. Unfortunately, the number and the variety of patients in many teaching hospitals across the country are unfavourable as far as undergraduates and postgraduates are concerned. I am sure this book will find a place in the armamentarium of all the students.



Dr Bhat is already the author of a popular textbook *SRB's Manual of Surgery*. I have no hesitation to believe that this book *SRB's Clinical Methods in Surgery* will be equally popular. Dr Bhat needs to write a book on operative surgery to complete that trilogy needed for medical students.

Dr CR Ballal
Professor Emeritus
Mangalore

Preface.....

It was my long time dream to bring out a good surgical clinical book for undergraduates, postgraduates and practicing surgeons. Even though I have written surgical manual for students of MBBS, Dental and Nursing category, I had an aspiration to write an adequate sufficient clinical book which can cover all category of students who need to learn clinical surgery properly. I have covered every aspect of clinical surgery with good illustrations in all chapters. This contains methods of basic clinical surgery for medical students. Clinical approaches, clinical analysis and clinical diagnosis with differential diagnosis are discussed in each chapter. However, therapeutic aspects, controversies and recent advances are not a part of this book. Students who want to learn in detail in these aspects are requested to refer to my textbook *SRB's Manual of Surgery*, 3rd edition or any other surgical books of their choice. I have taken enough care to discuss different clinical methods and signs. I have also referred standard clinical books and surgery books prior to writing this book. Many methods and signs which are old are well accepted but still many methods are controversial. But whatever given in this are commonly followed one; individual opinions and controversies not highlighted here.

I hope this book will be useful for all those who are keen to learn clinical surgery.

I sincerely appreciate everyone who has helped me. I thank Jaypee Brothers Medical Publishers, New Delhi for their support to bring out this book.

Any constructive criticisms are most welcome.

Sriram Bhat M

Acknowledgements.....

I am happy to bring out first edition of *Clinical Methods in Surgery*. This is due to constant help and support of many.

I thank our Chancellor, Dr Ramdas M Pai; Pro-Chancellor, Dr HS Ballal; Vice-Chancellor of MAHE, Dr Professor Rajashekar, Warrior; our beloved Dean, Professor (Major General Retd) Rajagopal MCh; our Vice-Deans, Professor Venkatraya Prabhu and Professor Urmila Kadilkar for their academic support.

I thank my beloved teacher, a great clinician and senior surgeon Dr Professor CR Ballal, Mangalore for accepting to give Foreword to this clinical book. I owe a lot to him.

I thank the Head of Department of Surgery, KMC, Mangalore, and Professor Thangam Varghese for her constant encouragement in academic work and progress.

I always remember my senior teachers, Professor CR Ballal and Professor Suresh Kamath for their constant help.

Our senior faculty in the department, Dr Professor K Prakash Rao who is a guide and encourager to everybody will be remembered as a special person by me always.

Surgical unit heads in our college Dr BM Nayak, Dr Anand Kni MCh, Dr Jayaram Shenoy, Dr Jayaprakash Rao, Dr Harish Rao, Dr Ramachandra Pai, Dr Alfred Augustine and Dr Shivananda Prabhu are always supportive for my work and are worth to be remembered always.

I am grateful to all my teachers and colleagues in surgery department who directly or indirectly helped me to bring out this book.

I appreciate District Medical Officer and Resident Medical Officer of Government Wenlock Hospital, Mangalore for their kind help.

I sincerely thank Professor Navinchandra Shetty, Head of Radiology Department, KMC, Mangalore and also other faculty of the department for their help in providing and guiding me in X-rays, CT scans and imaging methods.

I acknowledge Dr Professor Kishore Chandra Prasad, Head of Department of ENT for his help, guidance and encouragement in bringing out this book.

I thank Dr Shivaprasad Rai; Dr Yogish Kumar; Dr Ramesh; Dr Ashfaq Mohammed; Dr Kalpana Sridhar; Dr K Akbar; Dr Keshava Prasad; Dr Kishore Reddy; Dr Achaleswar Dayal; Dr Raghav Pandey; Dr Rupen Shah; Dr Ashwin Mallya; Dr Praveen; Dr Ashok Hegde; Dr Rajesh Ballal; Dr Devidas Shetty; Dr Venkatesh Sanjeeva; Dr Sunil; Dr Shanbogh; Dr Harish Nayak; Dr Subraya Kamath; Dr Venkatesh Shanbogh; Dr Shubanshu; Dr Chaithanya; Dr Nandakishore; Dr Shrishail; and Dr Sampath for their help in various aspects.

I sincerely appreciate Dr Raghavendra Bhat and Dr Ravichandra, Consultants in Radiology Department, Yenepoya Medical College, Mangalore for their contribution and affectionate help.

I thank Dr Ganesh Pai MCh (Paediatric Surgery); Dr Suresh Pai MCh; Dr Ashok Shetty (Cardiothoracic Surgery); Dr Muralidhar Pai MCh (Neurosurgery); Dr Sadashiva Rao, Paediatric Surgeon; and Dr Narayana Bhat, Paediatric Surgeon for their help and contributions.

I will never forget my close associates Dr Ganapathy MD, Mangala Hospital, Kadri, Mangalore and Dr Ashok Pandith MCh, Urologist for their affectionate help and encouragement in all my endeavours. They always stood with me in my difficulties.

I thank my friends Dr Manjunath Shetty, Urologist and Dr Jagadish MDS for their contributions to photos and X-rays and opinion on dental and maxillofacial topics. I thank Dr Balasaraswathy, Consultant Dermatologist, Mangalore for providing rare photographs.

My wife Dr Meera Karanth helped me day and night in editing this new book and without her help this could not have been possible. My beloved daughter Ananya helped me in drawing diagrams artistically. I enjoy her love and affection towards me.

I thank all my postgraduate students of Surgery Department (especially Dr Abhishek, Dr Ranjit, Dr Laxman, Dr Mahesh, Dr Adithya, Dr Nishyesh, Dr Solomon, Dr Sachin) who were helping regularly in bringing out this new book.

Words are not sufficient to remember all my patients who are the main material for the book. I pray for their good health always.

I appreciate Shri Jitendar P Vij, Chairman and Managing Director, Mr Tarun Duneja, Director (Publishing) and all staff of the Jaypee Brothers Medical Publishers (P) Ltd., New Delhi, for doing appreciable work in their respective field of printing and publishing. I also thank Mr V Venugopal, Branch Manager and staff of Bengaluru branch.

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1

Introduction on Clinical Examination

Introduction

Clinical examination is an art. It is an important basic essential part in surgical learning. Surgery is categorized as *clinical surgery*; *surgical principles and operative surgery*. So surgery is not just cutting. It involves proper clinical analysis; and application of principles in treating surgically related patients. All patients in the surgical ward need not undergo or need surgery. Conditions like cellulitis, amebic colitis or acute pancreatitis commonly does not require surgery but treated by surgeons. A surgeon should be a good clinician and physician all together to impart proper treatment to his (surgical) patients. Even though there are many sub-specialties in surgery now, basic clinical surgery remains the same. It is the pillar of surgical basis.

Two important parts in clinical methods are *symptoms and signs*. *Symptom* is the one patient complains of. It is the subjective sensation of the patient. *Sign* is the one which clinician elicits. It is an indication of existence of an objective evidence of a disease.

Clinician is the one who listens patiently; who sees carefully; who feels evidentially; who hears silently.

Clinical methods are schematically divided as:

History taking which is very important part. Careful detail history taking many times gives clue about the exact disease.

Physical examination includes general examination; inspection of the part (diseased or suspected) which is proper observation prior to palpation for specific findings; palpation is done once inspection is completed in detail; followed by percussion done in specific areas like abdomen and chest; later auscultation for altered or specific sounds in particular region.

Case taking or **Case analysis** includes:

- **Clinical methods.**
- **Clinical diagnosis.**

Investigations are done to come into final conclusion by various methods like X-ray, CT scan, ultrasound, blood tests and so on. Types of investigations are decided based on the clinical suspicion of the disease.

Final diagnosis is to plan the therapy, predict the outcome.

Treatment plan or protocol often differs for individual patient.

Postoperative/post therapy management.

Progress of the patient.

Follow-up after discharge and further treatment which is often needed after initial management.

History Taking

Clinician should spend adequate time for detailed history taking from the patient. If the patient is a child or patient is dumb, then history is given by the mother or close relative who takes care of the individual. Name and relation of the person who is giving history should be noted down. Patient should be made comfortable while taking history.

General History

Name

Correct name of the patient should be asked and noted down. It is better to remember the patients name while doing rounds at least up to the discharge of the patient. This helps to build a zone of comfort

with the patient. It may be helpful to keep a pocket note book to write down in short about details of the patient.

Age

Noting the age of the patient is important. Congenital anomalies occur in young age group. Cleft lip and palate; phimosis exists since birth. Branchial cyst even though of congenital origin occurs in later age group in 2nd or 3rd decade. Certain tumors like Wilm's tumor (kidney) and neuroblastoma occur in early childhood. Sarcomas develop in adolescents. Usually carcinomas occur after middle age. But malignancies can occur at any age group. Benign prostatic hyperplasia occurs in old age often causing retention of urine.

Sex

Certain diseases occur only in particular sex other than gender specific diseases. Hemophilia occurs only in males but females can be carriers. Thyroid diseases are more common in females. Carcinoma lung, stomach, kidney are more common in males but can occur in females.

Religion

Carcinoma penis is not seen in Muslims and Jews due to their religious practice of early circumcision in childhood. Duodenal ulcer perforation is common in Muslims during fasting month of Ramzan.

Residence, complete postal address and method of communication must be taken down: Many diseases have got geographical distribution. Hydatid disease is common in Australia, Iran, Greece, etc; Schistosomiasis is common in Egypt; Trypanosomiasis is common in Africa; amebiasis is common in tropical countries; filariasis is common in Orissa; leprosy in West Bengal; gallstones in Bihar and north east India; peptic ulcer in South India.

Occupation

Some diseases are common in people with certain occupations. Varicose veins are common in people who stand for long hours like bus conductors, garden workers, watchmen, traffic policemen, surgeons, and nurses, etc. Carcinoma urinary bladder is more com-

mon in workers in aniline dye factories. Sportsmen are more prone for injuries to ankle, knee and elbow.

Social status: Tuberculosis is common in low socioeconomic group; peptic ulcer disease is common in high socioeconomic group.

Social status is classified as Class I—professionals; Class II—Executive and higher management; Class III—Lower management and clerical; Class IV—Skilled laborers; Class V—Unskilled laborers.

Chief Complaints

Main complaints of the patient are mentioned in the order of occurrence. Complaints of same duration should be narrated in the order of severity. Example—Lump in the breast-6 months.

Ulcer in the swelling of breast-2 months.

Pain in the breast-1 month.

Fever-1 month.

Often proper leading questions are necessary to elicit clear-cut relevant history. But this should be used only after proper initial detailed history. History should be elicited in language which the patient is comfortable. One should not elicit diagnosis from the patient. Negative reply of the patient is also very relevant and so it should not be ignored.

History of Present Illness

It is detailed history in relation to onset of the present disease until date. It should be in order of occurrence. Each part of the history should be mentioned in detail before going to next part of the history.

Mode of onset of symptom: It may be gradual or sudden or initially slow but later progress rapidly. History suggestive of whether it is related to any trauma or any earlier disease should be asked.

Progress of the disease: Whether the symptoms are decreasing or increasing; gradual or rapid; or waxing and waning (increase-decrease-increase).

Past History

Earlier diseases should be detailed in order. Often patient may not know the name of the disease which he had earlier. History suggestive of specific disease should be elicited like tuberculosis, syphilis, leprosy,

bronchial asthma, diabetes mellitus, and tropical diseases. When such disease has occurred; detailed history of treatment taken; response to treatment should be asked for. Often patient might have got hospitalised for the treatment which should be asked in detail like place where he was hospitalised; duration; type of treatment (type of drugs, injections, etc). Earlier treatment summary/prescriptions if present should be taken and studied for reference. History of earlier surgery/trauma; its detail like duration of hospital stay, recovery period, any post-operative complications, drain placed or not, response of surgery whether patient is relieved of symptoms completely or partially, any operative notes available for reference should be asked.

Personal History

History of personal habits like smoking beedi or cigarettes with duration/frequency/number of beedi or cigarettes per day; history of drinking alcohol with duration, quantity, whether addicted, whether associated with alcohol induced problems should be noted.

Alcohol Intake

A *problem drinker* is one whose physical, social and mental well being is harmed by drinking. *One unit* of alcohol is 8 grams of alcohol in 290 ml of 4% beer. *Teetotaler* is one who has not taken alcohol in last one year. *Occasional drinker* is one who has not taken alcohol in last one month. *Light drinker* who drinks alcohol < 25 units per week in males; < 15 units in females. *Moderate drinker* who drinks 25-35 units/week in males; 15-25 units in females. *Heavy drinker* who drinks 36-50 units/week in males; 26-35 units in females. *Very heavy drinker* is > 50 units/week in males; > 35 units/week in females.

Smoking

Light smoker smokes one packet of cigarette/day for 2-10 years. *Moderate smoker* 1-10 packets of cigarettes/day. *Chronic heavy smoker* smokes 10-20 packets of cigarettes/day for 2-10 years

Type of diet is also important in relation to many diseases. It should be mentioned whether patient is married or not; number of children he/she has.

In females, detailed menstrual history should be noted. Time of menarche/menopause/regularity/presence of pain/dysmenorrhoea/white discharge/date of last menstrual period are noted in detail. Pregnancy history with number of pregnancies/abortions/normal delivery or Caesarean/last child birth should be noted.

Family History and Genetic History

Many diseases run in family. Examples are: piles; breast cancer; diabetes mellitus; tuberculosis, etc. If any of the family member is suffering from any disease; its detail, type, therapy for the same, whether he has undergone any surgery for the same and so on should be mentioned in detail. Number of siblings and their health details should also be taken.

Other Relevant History

In younger age group history of immunization for different diseases; history suggestive of allergy/reactions during earlier drug intake; history of long-term drug therapy like insulin, steroids, antidiabetics, antihypertensives, diuretics, hormones, etc. should be noted.

Pain

Pain is a commonest symptom which patient complains to a clinician. Latin word '*poena*' means penalty/punishment. Pain is the one patient feels; tenderness (sign) is the one surgeon/clinician elicits.

Types of pain:

Superficial pain: It is sharp usually localised pain, due to irritation of peripheral nerve endings in superficial tissues by chemical/mechanical/thermal/electrical injury.

Segmental pain: It occurs due to irritation of particular nerve trunk/root; located in particular dermatome of the body supplied by the sensory nerve trunk or root.

Deep pain: It is due to irritation of deeper structures like muscles/tendons/bones/joints/viscera. It is vague and diffuse when compared to superficial pain. It is often referred to common segmental areas of representation. Often spasm of skeletal muscle of same spinal cord segment can occur.

Psychogenic pain: It may be functional/emotional/hysterical.

Other types of pain: Like due to thalamic/spino-thalamic diseases/*causalgia* [intense burning pain along the distribution of the partially injured (and healed) nerve].

Specific points in history in relation to pain to be asked are: **original site of pain** is very important. In acute appendicitis original site of pain is in umbilicus; but later it shifts to right iliac fossa, i.e. shift of pain towards other site.

Time and mode of onset of pain: It is in sudden onset, rapidly progressive in acute appendicitis; it is of insidious onset and of long duration with episodic nature in chronic peptic ulcer; pain after trauma means very important and may be an emergency like internal organ injuries (liver, spleen, and kidney) or due to fracture bone.

Type/nature of pain: It may be superficial/deep; dull ache or sharp severe/pricking/bursting/vague aching (continuous mild pain), throbbing, scalding (burning sensation particularly felt during urination in cystitis, pyelonephritis, urethritis), pins and needles pricking sensation in peripheral nerve injury or irritation, shooting pain (seen in intervertebral disc prolapse and sciatica—pain shoots along the course of nerve), stabbing (sudden, severe, sharp, episodic—seen in perforated duodenal ulcer), distension pain (a feeling of restricted or distended like in paralytic ileus or intestinal obstruction), colicky pain is due to muscular contraction in a hollow tube in an attempt to obviate the obstruction by forcing the content out—gripping, episodic pain with vomiting and sweating (seen in intestinal colic, ureteric colic of stone, biliary colic of stone), twisting pain of bowel volvulus/twisted ovarian cyst/torsion testis, constricting pain around the chest by angina, etc.

Severity of the pain: Severe pain is common in acute appendicitis, acute pancreatitis, ureteric colic, perforation of bowel, acute peritonitis, intestinal obstruction, acute abscess.

Progression of pain: It may be persistent and progressive; or initially mild gradually increases, later subsides gradually; or fluctuates in intensity, i.e. increases and decreases in intensity at regular intervals or quickly reaches maximum and remains like that.

Duration of pain: Colicky pain lasts usually for a minute in each episode; anginal pain lasts for 3-5 minutes; an acute pain like of pancreatitis persists.

Periodicity of pain: Pain appears, persists for few weeks and then disappears for few weeks; again reappears. Such periodicity is often observed in chronic peptic ulcer; trigeminal neuralgia.

Precipitating/aggravating factors: Abdominal pain may get worsened by taking food like in gastric ulcer. Pain due to appendicitis, ureteric stone aggravates in change of position, walking, jolting. Pain of urinary bladder stone aggravates in standing position. In reflux oesophagitis pain increases while scooping. Pain in pancreatitis increases on lying down. Pain in intervertebral disc prolapse aggravates by lifting the weight.

Relieving factors of pain: Pain reduces by certain method and patient uses that method to relieve the pain. Hunger pain of early morning in duodenal ulcer is relieved by taking food. Pain of pancreatitis is relieved by sitting and bending forward. Propped up position relieves pain of reflux oesophagitis. In acute peritonitis, pain reduces temporarily by lying still.

Associated symptoms: Acute pain may be associated with pallor, sweating and vomiting. Migraine pain with vomiting and visual disturbances; intestinal/ureteric colic with sweating, vomiting and cold periphery; acute pyelonephritis and urinary infections with chills/rigors and fever; ureteric colic with haematuria; biliary colic with jaundice and pale stool are other examples of such association.

Time of occurrence of pain is often important in diagnosing the condition. In duodenal ulcer, hunger pain occurring in early morning or later evening is typical. Migraine occurs in early morning; frontal sinusitis induced headache occurs few hours after getting up.

Pain may move from one place to other.

Radiation of pain: It is extension of pain from original site to another site with persisting of pain at original site. This radiating pain is of same character of original site. Penetration of duodenal ulcer posteriorly causes pain both in epigastrium and back—is an example. Pain of pancreatitis radiates to back.

Referred pain: Pain is not felt at the site of the disease but felt at distant site. Diaphragmatic irritation causes referred pain at the tip of shoulder through same segmental supply of diaphragm (phrenic nerve C4, C5) and shoulder (cutaneous supply C4, C5). Hip joint pathology may cause referred pain in knee joint—through articular branches of femoral, obturator and sciatic nerves. Other *examples*—referred pain in ear from carcinoma tongue through lingual and auriculo-temporal nerve; referred pain in the epigastrium from the heart; referred pain in the abdomen from pleura; referred pain over the testis from the ureter.

Shifting/migration of pain: Origin of pain is in one site; later pain shifts to another site and pain at original site disappears. Pain when begins in viscera, is felt at the same somatic segmental area in the body; but once parietal layer is involved by inflammation/pathology pain is felt at the anatomical site. Example is pain of acute appendicitis, where the original visceral pain is at the umbilicus (T9 and T10 segments supply both umbilicus and appendix) which later shifts to right iliac fossa when once the parietal peritoneum of that area is inflamed.

Grading of pain is done using pain scale. It is compared to a 10 cm line numbered 0 to 10. This is called as *visual analogue scale* (VAS). Minimum is 0 means no pain. 10 is the worst excruciating pain. 2 is mild; 4 is discomforting; 6 is distressing; 8 is intense.

Vomiting

Vomiting is a common symptom heard in clinical practice. It may be due to—pregnancy, travelling sickness, labyrinthitis, gastritis, peptic ulcer, migraine, meningitis, intracranial tumour, ureteric colic, pyloric stenosis, carcinoma stomach (pylorus), intestinal obstruction, intracranial space occupying diseases, acute peritonitis, cholecystitis, pancreatitis, metabolic causes like diabetic ketosis, drug induced. Colour, quantity, smell of the vomitus should be found. Coffee ground coloured vomitus is seen in upper GI bleed. When blood comes in contact with gastric juice, hemoglobin forms acid haematin colouring contents blackish or dark brown. Vomitus may contain frank blood/clots. Presence of undigested material should be asked for. Oesophageal obstruction by achalasia cardia or stricture causes regurgitation. Nonbilious vomiting means obstruction proximal to sphincter of Oddi.

Bilious vomiting occurs in small bowel obstruction; which may be either yellow or green coloured. Faecal content in the vomitus suggests ileal/large bowel obstruction. Faeculent vomiting is also seen in gastroduodenal fistula. Content is brown in colour with faecal odour. Haematemesis should be distinguished from haemoptysis. Vomiting is *graded as follows*—None (0); one episode of vomiting in 24 hours (1); 2-5 episodes/24 hours (2); > 6 episodes/24 hours (3); needs parenteral fluid/nutrition (4).

Nausea

It is sense (feel) of vomiting. It may or may not end up with vomiting. It can be none (0); nausea present but able to eat (1); oral intake is reduced (2); No oral intake, on IV fluids (3).

Itching (Pruritus)

It is due to local or general causes. Multiple scratch marks are often obvious. It may be due—*Skin diseases*: urticaria, eczema, scabies (Psoriasis will not cause itching). *Local causes* contact dermatitis due to clothing, washing soap, washing powder infection from fungal, parasites like fleas, scabies; vaginal and rectal discharge. *Systemic causes* are obstructive jaundice due to bile acid irritation, Hodgkin's disease, leukaemia, uraemia, allergy/hypersensitivity, drug reactions, diabetes mellitus, etc.

Fatigue

It is subjective sensation of weakness (asthenia/lethargy). It is graded as none (0); fatigue over baseline (1); moderate fatigue (2); severe (3); bedridden (4).

Anorexia

Anorexia is loss of appetite. It is seen in anorexia nervosa, gastrointestinal cancers, tuberculosis, debilitating illness like sepsis. Anorexia is graded as none (0); loss of appetite (1); significant reduction in oral intake (3); unable to take orally requiring IV fluids (3). Satiety is sense of fullness after completion of meals. It is normal. *Early satiety* is a feature of GI malignancy.

Flatulence

Flatulence is frequent belching more than normal. *Regurgitation* is effortless return of food into the

mouth. It is associated with powerful involuntary contractions of abdominal muscles. It is seen oesophageal/OG junction obstructions like carcinoma and achalasia cardia. *Heartburn* is burning sensation behind the sternum due to acid reflux into the oesophagus.

Constipation

Constipation is defined as having bowel movement fewer than three times per week; with hard, dry, small sized stool; difficult to evacuate. It is graded as none(0); needs diet modification (1); needs laxatives (2); needs manual evacuation or enema (3); due to obstruction (4). Constipation can be *relative* wherein patient can pass flatus but not faeces; or *absolute* wherein patient neither can pass faeces nor flatus.

Diarrhea

Diarrhea is defined as more than 3 stools per day. It is usually soft, often foul smelling. Often it may be associated with incontinence. It is graded as increase of < 4 times/day (1); increase 4-6/day (2); increase > 7/day or with incontinence or need parenteral nutrition (3); needs intensive care with haemodynamic collapse (4).

Physical Examination

It should be done in privacy. Female patients should be examined in presence of a female/nurse. Examination should be done with limited clothing to elicit proper findings. Broad day light is ideal for examination. Usage of other lights may mislead or mimic some clinical findings like jaundice.

General Examination

This part of the examination is essential preliminary step in all patients.

Patient's intelligence level should be assessed while taking history. Uneducated people still can be intelligent.

Mental Status

Mental status and level of consciousness should be assessed in general but in particular in specific clinical situations like head injury, hepatic encephalopathy, septic shock, etc.

Grading of the mental status

Grade I	Properly oriented in time, space and person
Grade II	Conscious but without orientation of time, space and person
Grade III	Drowsy and semiconscious
Grade IV	Unconscious but responding to painful stimuli
Grade V	Unconscious and comatose and not responding to painful stimuli

Built and Nutritional Status

Built and nutritional status of the patient is important to be assessed. *Built* is structural organization of underlying skeleton. It is related to age and sex of the patient. *Gigantism* is height to that age is in excess than normal (in adult more than 6.5 feet). It may be racial; familial; endocrinal (hyperpituitarism, hypogonadism); genetic (Klinefelter's syndrome); metabolic (Marfan's syndrome, homocystinuria); overeating; cerebral causes. *Dwarfism* is height to that age and sex is far less than normal (below 4.5 feet). It can be hereditary, chromosomal (Turner's syndrome, Down's syndrome); delayed growth; nutritional (Rickets); endocrinal (hypopituitarism, hypothyroidism, excess androgens, congenital adrenal hyperplasia, insulin insufficiency); skeletal (achondroplasia, spinal deformities); systemic diseases (uraemia, cyanotic heart diseases, cirrhosis). In *normal adult*, height of the person is equal to length of arm span. Upper segment from vertex to pubic symphysis is equal to lower segment from pubic symphysis to heel. In infants upper segment is more than lower segment and height is more than arm span. This *infantile body frame* persists in achondroplasia, cretinism, and juvenile myxoedema. Greater arm span than height and greater lower segment is observed in Marfan's syndrome, homocystinuria, Klinefelter's syndrome, Frohlich's syndrome.

Nutrition is the proportion of soft tissue structures (muscles, soft tissues, fat) in relation to the bony structure. In gastrointestinal malignancies or in other malignancies with metastases patient will be cachexic. Protein deficiency causes rough skin, brittle hair, and oedema feet. Fat deficiency causes cachexia, hollow cheeks, and loss of fat in hips, abdomen and subcutaneous tissues of elbow. Deficiency of minerals and vitamins has got specific features.

Weight Gain

Weight gain is increase in weight. It is graded as increase of < 5% (0); increase of 5-10% (1); 10-20% (2); > 20% (3). It is seen in obesity, pregnancy, myxoedema, water retention, Cushing's syndrome.

Weight Loss

Weight loss is graded as loss of < 5% (0); 5-10 % (1); 10-20% (2); > 20 % (3). But time duration of weight loss is also important. *Definition of significant weight loss* (2009): Weight loss more than 5% (up to 7.5%) in 30 days; weight loss more than 7.5% (up to 10%) in 60 days; weight loss more than 10% in 180 days.

Wasting

It is obvious on the upper half of the body as there is often oedema due to hypoproteinaemia in lower half of body. By looking at the shoulder girdle, loose skin of arms, trunk and buttocks, severity of wasting can be assessed (**Fig. 1.1**). It is observed in starvation, severe gastroenteritis, tuberculosis, anorexia nervosa, diabetes mellitus, advanced carcinomas, gastrointestinal malignancies, and old age.



Fig. 1.1: Ascites with wasting proximal part probably due to malignancy.

Malignant Cachexia

Malignant cachexia is emaciated (**Fig. 1.2**), languid, shallow, pale face, loose wrinkled dry skin, loss of fat, lost appetite/weight/energy with oral infection. Profound loss of weight is typical.



Fig. 1.2: Typical malignant cachexia.

Attitude

Attitude of the patient in the bed is good thing to observe. Comatose patient/paraplegic or quadriplegic is silent and immobile. Patient in shock or with peritonitis may not move due to pain. Patient with ureteric stone may be restless and rolling in the bed due to severe colicky pain. Position of the patient in the bed is called as *decubitus*. It is often typical in certain diseases like cerebral irritation, cerebral palsy, etc. In hemiplegia patient lies with one side immobile, with affected arm flexed and legs externally rotated and extended. In tetanus, patient develops stiff neck. In ureteric colic, patient is restless with rolling and tossing over the bed. In acute peritonitis patient lies in the bed still and motionless. In cardiac diseases, patient is comfortable in sitting up position. In pneumonia, patient lies on the affected side to make that side immobile and restricted so as to reduce the pain.

Stature is the total height from vertex to soles. **Posture** is positional relationship of different regions of the body. *Normal posture* is—moderate lordosis of cervical and lumbar spine; kyphosis of thoracic and sacrococcygeal region; forward pelvic inclination 30°; normal rotation of femur; line from the mastoid down passes through the middle of the shoulder and hip, anterior to knee and lateral malleolus.

Face Look

Typical face is diagnostic of some diseases. *Hippocratic facies* is seen in generalised peritonitis. Face with typical pale look is seen in chronic renal failure, *risus sardonicus* in tetanus; *mask face* in Parkinsonism;

moon face in Cushing's syndrome is to be noted. *Acromegaly* (due to increased growth hormone in pituitary acidophilic adenoma) shows large face due to overgrowth of soft tissues in face, nose, tongue, air sinuses; large hands (due to enlargement of bones of distal phalanges)—*facies of Punch* or '*Punch and Judy*' or an '*Ape man*'. Skin is greasy; mental acumen is normal (in myxoedema skin is dry with decreased mental acumen). In *scleroderma*, progressively thickened, pale, waxy skin with reduced facial expressions, microstomia, telangiectases on cheeks, mouth and nose, with fine white horizontal scars in the neck in transverse skin creases (with oesophageal stenosis and vasculitis) are seen. In *Myasthenia gravis* weakness of all muscles is found; in particular of eyelids showing drooping of eyelids with weakness of face muscles and jaw (**Fig. 1.3**). *Cretin* is a neonate with deficient thyroid hormone (cured by thyroid hormone supplement); diagnosed at birth; with broad flat face, wide apart eyes, protruded tongue. *Down's syndrome/Mongolism* is a congenital abnormality with extra-chromosome 21 and total chromosomes 47 (instead of 46); males and females and all races are equally affected. Features are—mental retardation, floppiness, short stature, outer ends of the palpebral fissures slanted upwards with prominent epicanthic folds, flat face, protruded tongue and squint.

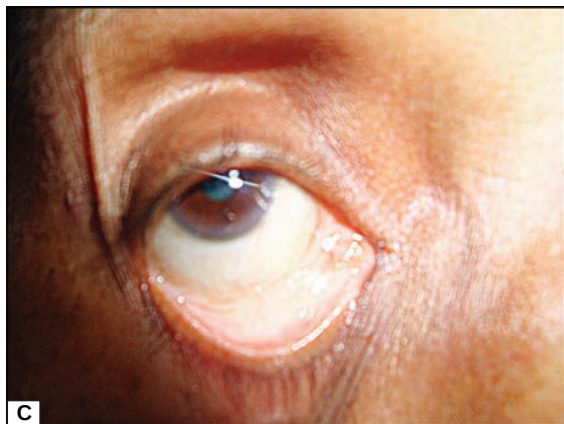
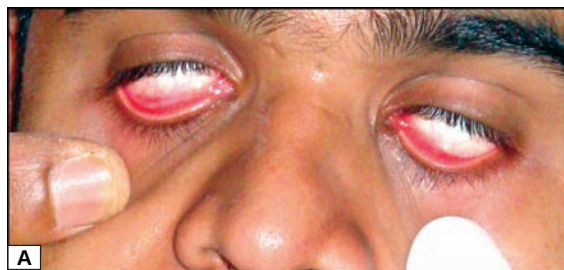
Klinefelter's syndrome is a congenital abnormality in a male having XXY chromosomes instead of normal XY chromosome. Patient is tall, with female distribution of fat around breast and pelvis but normal hairs in face and pubis. Patient is having small testis without sperms. *Turner's syndrome* is a congenital abnormality of female, having only one X chromosome, XO instead of XX. Short, webbed shoulder, widened neck with prominently running skin fold from neck to shoulder—are typical.



Fig. 1.3: Eyes and face should be examined carefully as part of general examination. Note the visible lower sclera—could be due to exophthalmos.

Pallor

Pallor is checked in lower palpebral conjunctiva, mucous membrane of lips and cheeks, nailbeds and palmar creases. Causes for pallor are—anaemia, massive bleeding, shock and anxiety status (**Figs 1.4A to C**).



Figs 1.4A to C: Lower eyelid is retracted to see the conjunctiva for pallor. Note the normal conjunctiva and conjunctiva with pallor.

Cyanosis

It is due to rise in level of reduced haemoglobin in the blood causing blue/purple discolouration in the skin and mucous membrane. A minimum of 5 gm/dl of redu-

ced haemoglobin should be present in the circulation to cause cyanosis. So in severe anaemia (Hb% below 5 gm %), cyanosis is not seen. Two types of cyanosis are observed—*peripheral and central*. Peripheral cyanosis is due to poor perfusion of peripheral vessels causing reduction in oxyhaemoglobin in the capillaries. It is seen in peripheral vasoconstriction due to any cause like exposure to cold temperature, reduced cardiac output, profound shock where blood is diverted from periphery to vital organs like brain, liver, and kidney. Peripheral cyanosis is checked in nailbed, palm and toes, tip of the nose. Here limb is cold and inhaling pure oxygen may not reduce it. Tongue is not involved in peripheral cyanosis. Central cyanosis occurs due to reduced oxygen saturation of arterial blood due to poor oxygenation in the lungs. It may be due to congenital heart disease with left to right shunt (cyanotic heart disease), congestive cardiac failure, lung diseases, and high altitude due to low oxygen partial pressure. Limb temperature is normal in this type. Clubbing and polycythaemia is common here. Pure oxygen inhalation reduces the central cyanosis. It is confirmed by checking in tongue (**Fig. 1.5**), nailbed, palms and toes. Methaemoglobinaemia or sulphaemoglobinaemia (abnormal pigments) also causes cyanosis but with

normal arterial tension. In carbon monoxide poisoning, carboxyhaemoglobin prevents reduction of oxyhaemoglobin and so there will not be any cyanosis but cherry red discoloration develops.

Differential cyanosis: Patent ductus arteriosus (PDA) with reversal of shunt causes only lower limb cyanosis. PDA with reversal of shunt with transposition of great vessels causes only upper limb cyanosis. PDA with reversal of shunt with preductal coarctation of aorta causes cyanosis of left upper limb and both lower limbs.

Polycythaemia

Polycythaemia is excess of circulating red blood cells giving patient a purple-red florid appearance; it heightens the colour of all the skin, cheeks, neck, backs of hands and feet whereas cyanosis is limited to tips of hands, feet and nose.

Jaundice

Jaundice is yellowish discoloration of skin and mucous membrane. Tissues and body fluids are also discoloured yellow. Bilirubin has more affinity to elastic tissue, blood vessels and nervous tissue. So it is better seen in sclera and skin. During recovery, bilirubin takes longer time to get cleared from elastic tissue and so clinical jaundice persists for little longer time than biochemical disappearance of jaundice. Initially it is pale lemon yellow colour, later gets darkened becomes yellow-orange, olive greenish yellow as seen in obstructive jaundice. Jaundice is due to deposition of bile pigments with excess of it in plasma. It is checked in upper sclera (better seen against white background; by asking the patient to look at his feet and clinician pulls the upper eyelid upwards). It also can be checked in *nailbed, ear lobule, nasal tip, and on under surface of tongue*. Greenish colour is due to deposition of biliverdin. Scratch marks observed on the dorsum of the body (forearm, neck, back) is due to deposition of bile acids which releases excess histamine causing itching (**Figs 1.6A to C**).

Jaundice may be due to *pre-hepatic* cause (excess haemolysis); *hepatic* (liver dysfunction—hepatitis, sepsis, drugs, cirrhosis); *post-hepatic* (CBD stones, carcinoma pancreas, drugs—obstructive); *congenital*



Fig. 1.5: Central cyanosis is checked in the tongue-dorsum.



Figures 1.6A to C: Janudice is checked in sclera by asking the patient to look down on the feet and examiner pulls the upper eyelids upwards. It is also checked in nasal tip, ear lobule, fingertips and under surface of the tongue.

hyperbilirubinaemia (*Gilbert's syndrome* causing altered bilirubin transport and so increase in unconjugated bilirubin; *Crigger-Najjar syndrome* causing disturbance in bilirubin conjugation and so increase in unconjugated bilirubin; *Dubin-Johnson syndrome* and *Rotor's syndrome* causing disturbance in excretion of bilirubin and so increase in conjugated bilirubin). Aged red cells get lysed in the reticuloendothelial cells and breakdown into haem and globin. Haem is divided into globin and bilirubin. Bilirubin is combined with albumin and transported to liver. In the liver bilirubin get separated from albumin and is conjugated to bilirubin glucuronide by glucuronyl transferase. This conjugated bilirubin glucuronide is water soluble and can be excreted in kidney (So in

obstructive and hepatic jaundice bile pigment-bilirubin is seen in the urine). This conjugated bilirubin is excreted through biliary canaliculi reaching intestine. In the intestine, it is converted into stercobilinogen and urobilinogen by intestinal bacteria. 70% of this is absorbed in the colon and brought back to liver as enterohepatic circulation (**Fig. 1.7**). Unabsorbed stercobilinogen colours faeces brown. Circulating urobilinogen is taken up by kidneys for excretion. If direct bilirubin in the serum is more than 0.4 mg%, then bilirubin is seen in urine. Normal urinary urobilinogen is 100-200 mg/day. It is absent in obstructive jaundice. Normal faecal stercobilinogen is 300 mg/day. It is also absent in obstructive jaundice.

Hypercarotinaemia

Hypercarotinaemia mimics jaundice which is due to increased yellow pigment carotene. It is seen equally in face, palm, sole and skin but not seen in sclera. It is common in vegetarians who eat more raw carrot. Mepacrine therapy also causes yellow discolouration.

Pigmentation

It is usually an increase in natural brown pigmentation of the skin. Often pigmentation by other colours like blue/red also can occur. Pigmentation can be generalised or localised.

Generalised: It occurs in Addison's disease (seen in skin and buccal mucosa); arsenic/silver poisoning; haemochromatosis; Gaucher's disease.

Localised: It occurs in pregnancy (around areola, midline abdomen); venous diseases of lower limb (medial third of leg and ankle); *erythema eb agne* (in the exposed part of leg); ultraviolet and high voltage irradiation; *café au lait* spots of neurofibromatosis; naevi; melanomas; pellagra (nicotinic acid deficiency); hyperthyroidism (bronzing of eyelids); rheumatoid arthritis.

Examination of Nails

A transverse groove (transverse lines/Bean's lines) seen at similar levels of each nails is suggestive of

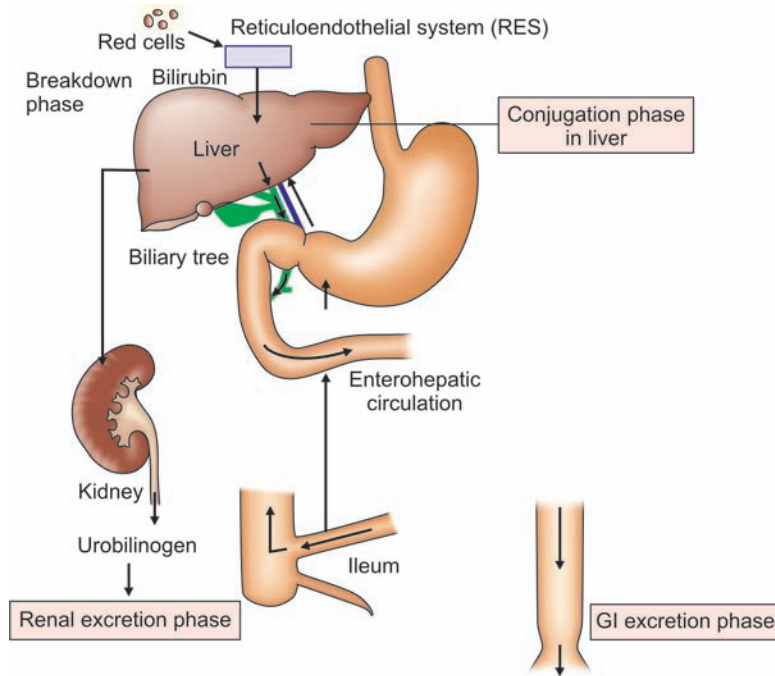


Fig. 1.7: Enterohepatic circulation.

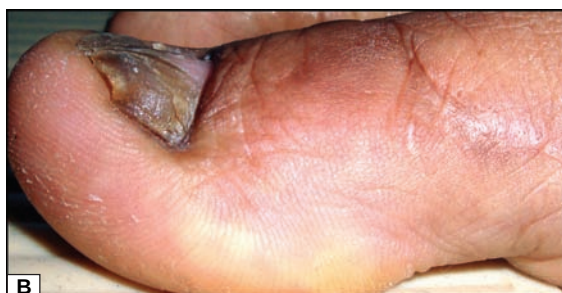
systemic disease/general debilitating illness. Pallor can be seen in nailbed. In iron deficiency anaemia (*Plummer-Vinson syndrome*) nails may be brittle/flat (*platynychia*)/spoon shaped (*koilonychia*). Splinter haemorrhages are seen in nailbed in bacterial endocarditis and bleeding disorders. Discoloured, deformed, pitted nails are seen in psoriasis. Hypoalbuminaemia causes whitening of the nailbed—*Terry's sign*. *Onychia* is deformity of the nail—seen in fungal infection or tuberculosis. Specific discolourations are seen in Raynaud's disease, silver and mercury poisoning. Ribbing, brittleness, falling of nails are seen in syringomyelia, leprosy and tabes dorsalis. Nailbed infarcts are seen in vasculitis due to SLE or polyarteritis. *Onychogryphosis* (in toe) is heaping up of nail and curling over the end of the toe due to failure of normal sliding mechanism of the nail and is due to trauma or old age. *Ingrowing toe nail* is common in margins of the nail of great toe where irregular edge of the nail grow beneath the lateral nail fold due to improper trimming of the nail causing repeated pain and infection (**Figs 1.8A to 1.10B**).



Figs 1.8A and B: Nails should be examined in both hands and feet (fingers and toes) for change in colour, splinter haemorrhage, clubbing, pallor, koilonychia and other features.



Figs 1.9A and B: Changes in the toe nail also should be observed. Note the pallor and koilonychia in the toe nails.



Figs 1.10A and B: Note the change in the great toe nail. It could be onychogryphosis.

Clubbing

It is bulbous enlargement of the soft parts of the terminal phalanges with both transverse and longitudinal curving of the nails. It is due to interstitial oedema and dilatation of the arterioles and capillaries. There is loss of normal angle between surface of the nail and the skin covering the base of the nailbed. When a normal nail is viewed from side, plane of the nail and the plane of the skin covering the base of the nailbed form an angle of 130° – 170° (Lovibond angle). In clubbing tissue hypertrophy beneath the nailbed makes the base of the nail bulge upwards distorting the nail growth causing nail to be curved in both directions. So in clubbing plane of the nail and plane of the skin covering the nailbed form an angle which is greater than 180° (**Figs 1.11A to D**).

Causes: It can be due to pulmonary (Carcinoma bronchus, lung abscess, bronchiectasis, tuberculosis with secondary infection); cardiac (cyanotic congenital heart disease, infective endocarditis); gastrointestinal (ulcerative colitis, Crohn's disease, cirrhosis); endocrinal (myxoedema, acromegaly, exophthalmic ophthalmoplegia—*thyroid acropachy*); other causes (hereditary, idiopathic), *unilateral* in Pancoast tumour, subclavian/innominate artery aneurysm: *unidigital* in trauma or tophi deposition in Gout, only in upper limbs in heroin addicts due to chronic obstructive phlebitis.

Grading:

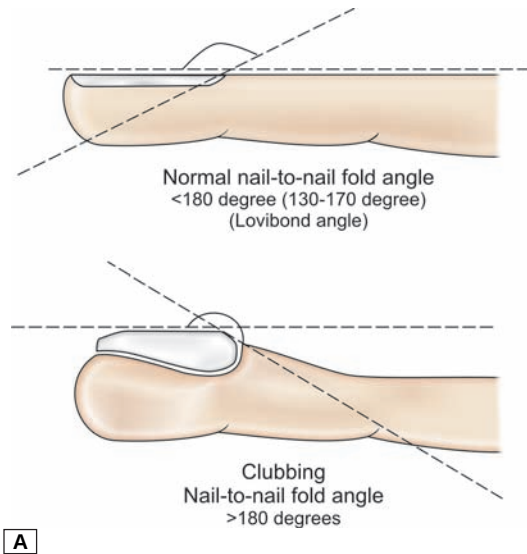
Grade I: Softening and fluctuation of nailbed;

Grade II: Obliteration of angle of the nailbed with loss of longitudinal ridges and formation of convexity from above downwards and side-to-side;

Grade III: Swelling of the subcutaneous tissue over the base of the nail causing overlying skin tense, shiny and wet increasing the nail curvature;

Grade IV: Swelling of the fingers occurs in all dimensions, associated with hypertrophic pulmonary osteoarthropathy causing pain and swelling of the hand and radiographic features of subperiosteal new bone formation.

Disappearance of diamond shaped gap between nails when fingers are apposed—*Schamroth's sign*; **Pathogenesis:** Hypoxia leads to opening up of deep arteriovenous fistulas which increase the perfusion of the fingers and toes causing its hypertrophy. It may be due to reduced venous blood ferritin which escapes oxygenation in the lungs, which after entering



A



C



B



D

Figs 1.11A to D: Typical clubbing. In normal individual angle from skin to nail fold is 130 to 170 degree. In clubbing it is more than 180°. In clubbing both longitudinal and transverse curvatures are increased.

the systemic circulation stimulates dilatation of arteriovenous anastomosis leading to hypertrophy and clubbing of terminal phalanx. **Pseudoclubbing** is seen in hyperparathyroidism due to undue bone resorption resulting in disappearance of terminal phalanges causing telescoping of soft tissues into the terminal phalanges which appears like clubbing. Nail is not having curvatures here.

Oedema

It is the collection of fluid in the interstitial spaces or soft tissues. Oedema will be clinically evident only when fluid accumulates more than 5 litres. Pitting on pressure occurs only when circumference of the limb is increased by 10% (**Figs 1.12A and B**).

Mechanism: Fluid accumulates in the interstitial space following— Increased capillary permeability like in acute inflammation (cellulitis); increased capillary pressure (cardiac failure); decreased osmotic pressure (hypoproteinaemia); lymphatic block (filariasis). Pitting on pressure is the cardinal sign of oedema. Using pulp of the finger/thumb firm pressure is applied for few seconds over the skin on a bone surface like lower part of medial aspect of leg just above the malleoli. Indentation or pitting is seen on releasing the finger. Slow reaccumulation of fluid in few minutes is observed. Nonpitting oedema is observed in late stage of lymphoedema. Oedema is commonly observed in most dependent part—lower limbs. In bedridden patient, it may be seen on sacral region. Often limb



Figs 1.12A and B: (A) Pitting oedema in the leg. (B) Note oedema with multiple ulcers.

oedema may also be associated with ascites or pleural effusion. Oedema can be generalised or localised. Generalised oedema is called as *anasarca*. It is due to cardiac, renal, hepatic or nutritional causes. Localised oedema is due to cellulitis, lymphatic causes, venous diseases, pretibial myxoedema of thyrotoxicosis. *Causes* may be classified as bilateral (cardiac, renal, hepatic, IVC obstruction, allergic, nutritional, toxic) or unilateral (lymphatic, traumatic, infection, metabolic like gout, DVT/varicose veins, hereditary). In CCF oedema is in most dependent position—in lower limbs and is more in evening. In LVF pulmonary oedema develops earliest and so dyspnoea, basal crepitations, cough are typical. In pericardial effusion, lower limb oedema, ascites, hepatomegaly—soft smooth liver, raised JVP without pulmonary oedema is observed. In renal cause oedema develops first in eyelids and face, and then it becomes generalised into legs and ascites. In hepatic cause like portal hypertension, ascites develops first due to increased portal pressure and hypoproteinaemia, and then lower limb oedema develops. In myxoedema, oedema is nonpitting. Here oedema over the lateral aspects of the eyelids is typical.

Oedema grading—None (0); asymptomatic, not requires drug therapy (1); symptomatic requires drug therapy (2); symptomatic, with limited function, not responding to therapy (3); anasarca (4).

Visible Veins

Patient should be examined for visible veins. With normal venous pressure external jugular vein is invisible or just visible for short distance. Raised venous pressure causes engorgement of external jugular vein. Bilateral engorgement of external jugular vein/neck veins may be due to myocardial infarction or intravenous fluid infusion or retrosternal goiter/thoracic outlet obstruction. Unilateral engorgement of vein is due to compression by lymph nodes, tumour. In toxic goiter neck veins may be prominent due to increased vascularity. In SVC obstruction, inguinoaxillary veins, chest wall veins, neck veins may be prominent with flow of blood from above downwards and through groin veins (across watershed area) to IVC. In IVC obstruction, veins in the flanks (both sides) will be prominent, with direction of flow from below upwards towards axillary vein along inguinoaxillary vein. Unilateral such flow is observed in unilateral blockage of common or external iliac vein. IVC obstruction is *classified as*—below the renal vein (standard presentation); *at the level of renal vein* (lumbar pain, haematuria, proteinuria); *above the level of renal vein* (like Budd-Chiari syndrome). *Caput medusae* is visible dilated veins radiating from umbilicus, seen in portal hypertension (**Figs 1.13A to 1.14B**).



A



B



C



D

Figs 1.13A to D: Superior vena caval obstruction causing dilated veins in the chest wall. Note the direction of flow from above downwards towards lower abdomen and to inferior vena cava.



Figs 1.14A and B: Dilatation of abdominal veins including inguinoaxillary vein and bilateral varicose veins in a patient with IVC obstruction.

Jugular Venous Pulse (JVP)

Normal JVP has got 3 positive waves a, c and v and 2 negative waves x and y. 'a' wave is due right atrial contraction (**Fig. 1.15**). It is absent in atrial fibrillation; prominent in tricuspid/pulmonary stenosis. *Cannon a wave* is seen in complete heart block, ventricular

tachycardia. 'c' wave is due to carotid artery impact into jugular vein and right ventricular systole. 'x' wave is due to fall in right atrial pressure and atrial relaxation. It is absent in tricuspid regurgitation. It is prominent in constrictive pericarditis. 'v' wave is due to right atrial filling. Giant v wave is seen in tricuspid regurgitation. 'y' wave is due to opening of tricuspid valve causing rapid inflow of blood from right atrium into the right ventricle. Rapid y descent occurs in constrictive pericarditis, heart failure and tricuspid regurgitation. Jugular venous pressure is 3-4 cm of water. It is elevated in cardiac tamponade, right ventricular failure, tricuspid stenosis, increased blood volume, asthma, emphysema, SVC obstruction. It is reduced in shock, dehydration. During normal inspiration, intrathoracic pressure falls and venous blood flow to thorax increases causing inspiratory collapse of jugular venous pressure. In constrictive pericarditis when intrapericardial pressure rises, there will be paradoxical increase in jugular venous pressure during inspiration—*Kussmaul's sign*.

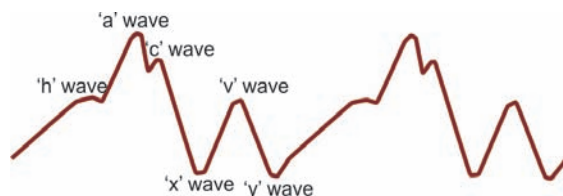


Fig. 1.15: Normal jugular venous pulse-waves.

Pulse Means Arterial Pulse

Pulse is an ideal indicator of severity of many diseases. It is increased in sepsis, severe pain, shock, fever, toxic thyroid. It is also altered in all cardiac conditions. Rate (count the pulse); rhythm (regularity); tension and force; character; condition of arterial wall should be noted. Pulse felt usually is radial pulse (against head of the radius) but when indicated, other pulses in the body also should be examined. It is felt using three fingers-index, middle and ring. Ring finger is kept distally to obliterate the retrograde pressure transmission; middle finger is over to feel the pulse; index finger is kept proximally to control and fix the artery to reduce the blood flow while checking the vessel wall thickness. *Pulse is counted for full one minute. Counting for few seconds and multiplying is wrong. Normal pulse has got a*

small anacrotic wave in the upstroke (which is not felt), a big tidal percussion wave which is felt (**Fig. 1.16**). During downstroke there is a dicrotic notch with a dicrotic wave (both are not felt). *Anacrotic wave pulse* is felt in severe aortic stenosis. *Pulsus bisferiens* is rapid rising, twice beating waves in the systole of the pulse; felt in idiopathic hypertrophic subaortic stenosis, severe aortic incompetence with mitral stenosis. *Dicrotic pulse* is twice beating pulse with initial normal percussion wave of systole and eventual abnormal prominent dicrotic wave in diastole. It is seen in reduced peripheral resistance like CCF, cardiac tamponade, typhoid fever. *Pulsus alterans* is alternate strong and weak beats; due to alternate contractions of the cardiac muscle; seen in left ventricular failure, toxic myocarditis. *Pulsus paradoxus*—During inspiration there is increased venous return to right atrium; lung expansion causes pooling of blood in the pulmonary vessels causing decreased venous return to left atrium and ventricle. It causes decreased left ventricular output and arterial pressure during inspiration by 3-10 mm Hg. When this fall in systolic pressure is exaggerated more than 10 mm Hg, it is called as pulsus paradoxus. It is seen in SVC obstruction, airway obstruction, asthma, pericardial effusion. In immobile thoracic cage pulsus paradoxus does not exist. *Pulsus bigeminus* with coupling occurs in atrioventricular block. Thready pulse is rapid, small waved pulse seen in shock, cardiac diseases. *Waterhammer pulse* is large bounding pulse with a forcible jerk, disappearing quickly. It is due to sudden fall in peripheral resistance; seen in thyrotoxicosis, AV fistula, beriberi, aortic regurgitation, PDA. Tachycardia means increased pulse rate more than 100/minute. Bradycardia (Greek-slow) is decreased pulse rate less than 60/minute.

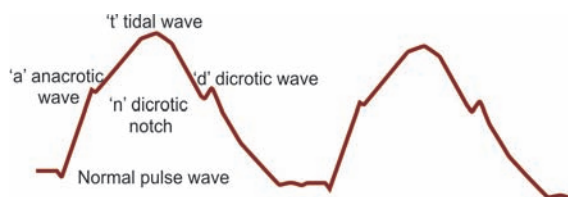


Fig. 1.16: Normal arterial pulse wave.

Blood Pressure (BP)

BP is essential part of the general examination in all cases. It gives the idea about the general condition of the patient along with other parameters. BP is lateral pressure exerted by the column of blood on the walls of the arteries. Systolic pressure is due to stroke volume of the heart and stiffness of vessels. It is the maximum pressure produced during (cardiac cycle) systole. Diastolic pressure is due to peripheral resistance. BP varies in phases of respiration. It is the minimum pressure exerted during cardiac cycle (diastole). It is related to emotion, exercise, smoking, alcohol, tobacco, meals, temperature, anxiousness, circadian rhythm, age, race, obesity, etc. BP is recorded by indirect method. *Riva Rocci* invented *sphygmomanometer*. It contains mercury manometer, cuff and air pump. Russian surgeon *Korotkoff* originated the method of placing of stethoscope over cubital fossa to hear sounds of brachial artery. Procedure of taking BP should be meticulous. Patient should be explained about the procedure. Patient should be on rest for 5 minutes prior to checking BP. Patient should avoid exertion or meals 30 minutes prior to checking BP. Clothing of the arm should be removed or kept as it is without folding (folding may cause constriction band). Width of the inflatable bladder cuff should be about 40% of the upper arm circumference (12-14 cm width in average adult); length of the inflatable bladder should be 80% of upper arm circumference, almost long enough to encircle the arm. Standard commonly used is 12 × 23 cm size. In the thigh 18 × 24 cm is used. In obese, 12 × 35 cm sized cuff is used. In children smaller sized cuff (width 3 cm in infants; 8 cm in children) is used. Bladder of the BP cuff should encircle the arm completely; center of the bladder cuff should be over brachial artery; ideally rubber tubes should be placed on the inferior aspect in the line of the brachial artery (eventhough tubes are commonly placed superiorly to make stethoscope placement over cubital fossa easier); though bell of stethoscope gives better sound; diaphragm of the stethoscope is commonly used due to its ability to cover wider area and easier to secure. Usual position is supine lying down with arm supported to heart level. In sitting/standing position arm should be horizontal at 4th intercostals space of the sternum. If arm is not

supported, arm with isometric contraction will elevate the diastolic BP by 10%. In normal individual, there is not much difference in BP in standing, sitting or lying down positions. BP in right arm is higher by up to 10 mm Hg; if BP is higher by more than 10 mm Hg then it should be analyzed carefully. Repeat inflations of cuff will raise the systolic and diastolic BP and give false readings. So cuff should be inflated rapidly and deflated early and completely; further repeat readings are taken with a gap of 15 seconds.

Phases in BP measurement: *Phase I:* Appearance of faint clear tapping sound which gradually increases in intensity; *Phase II:* Softening or swishing sounds; *Phase III:* Return of sharper crisper sounds; *Phase IV:* Soft, blowing, muffling of sounds; *Phase V:* Disappearance of sounds completely. Phase I is systolic BP; Phase V is diastolic BP. *Hypertension* is persistent raised systolic (above 140 mm Hg) or diastolic (above 90 mm Hg) BP. It is sustained elevation of systemic arterial pressure. It could be—essential HT; renal; vascular; endocrinal; neurological; haematological. *Hypotension* is diminished BP (systolic pressure less than 90 mm Hg). It could be due to—postural, cardiac, endocrinal like Addison's disease; tuberculosis, malignancy, dehydration, shock, haemorrhage, hypovolaemia, anaemia, anorexia nervosa.

Respiration

Tachypnoea is rapid breathing seen in fever, shock, hypoxia, acidosis, tetany, hysteria. Gradual deepening of respiration alternating with short periods of apnoea is called as *Cheyne-Stokes respiration*.

Fever/Rise in Temperature

Normal body temperature is balance between heat gain and loss maintained by hypothalamus. It is the temperature of viscera and body tissues. Normal temperature is 36.7°C–37.5°C (98 to 99°F–98.6°F). A diurnal variation of 1°C is normal; lowest temperature is during morning 2–4 AM highest being in afternoon. *Fever* is increase in body temperature more than 1°C or more than the maximum range. Hypothermia is 35°C/95°F or below; subnormal temperature is 35°–36.7°C/95°–97°F; normal is 36.7–37.5°C/98° to 99°F (98.6°F); mild fever is 37.2°–37.8°C/99°–100°F; moderate fever is 37.8°–

39.4°C/100°–103°F; high fever is 39.4°–40.5°C/103°–105°F hyperpyrexia is more than 40.1°C/106°F.

Types of fever:

Continuous fever: Fever persists throughout the day and does not fluctuate more than 1°C in 24 hours. It is seen in pneumonia, urinary infection, endocarditis.

Remittent fever: Fever is above normal throughout the day but there is fluctuation of more than 1°C in 24 hours. *Intermittent fever*—temperature is present only few hours a day and reaches to normal. It is observed in malaria, Kala azar. When fever develops daily, it is called as *quotidian*; when fever develops on alternate days it is called as *tertian*; when it occurs every third day it is called as *quartan*.

Pel-Ebstein fever: Recurrent bouts of fever and afebrile periods occur at regular alternations. Fever rises for 3 days, remains high for 3 days, remits in 3 days and goes for an afebrile period of 9 days to develop fever again in the same manner. It observed in brucellosis; earlier also thought to be due to Hodgkin's lymphoma.

Fever with chills is sensation of cold with fever. *Rigor* is profound chill with piloerection (*gooseflesh*) with teeth shattering and shivering (**Fig. 1.17**). *Pyrexia Unknown Origin (PUO)* is defined as—fever more than 101°F; more than 3 weeks of duration; failure to reach into a diagnosis even after one week of inpatient investigation.

Causes for fever: Infective (bacterial, viral, fungal, parasitic); neoplastic; vascular (myocardial infarction, pulmonary embolism, pontine/subarachnoid haemorrhage); traumatic; collagen diseases; endocrinal; metabolic (Gout, acidosis); haemolytic.

Grading of fever: None (0); 38–39°C (1); 39.1–40° (2); > 40° for 24 hours (3).

Tongue

Tongue may be large called as *macroglossis*. It is seen in lymphangioma, haemangioma, acromegaly, myxoedema, cretinism, amyloidosis. *Tongue tremor* is observed in thyrotoxicosis (primary). It is checked with tongue kept inside the oral cavity. If tongue is protruded, tongue twitchings may mimic tremor. Tongue is bright red in colour normally—due to rich blood supply through capillary network. Pallor is seen in anaemia,

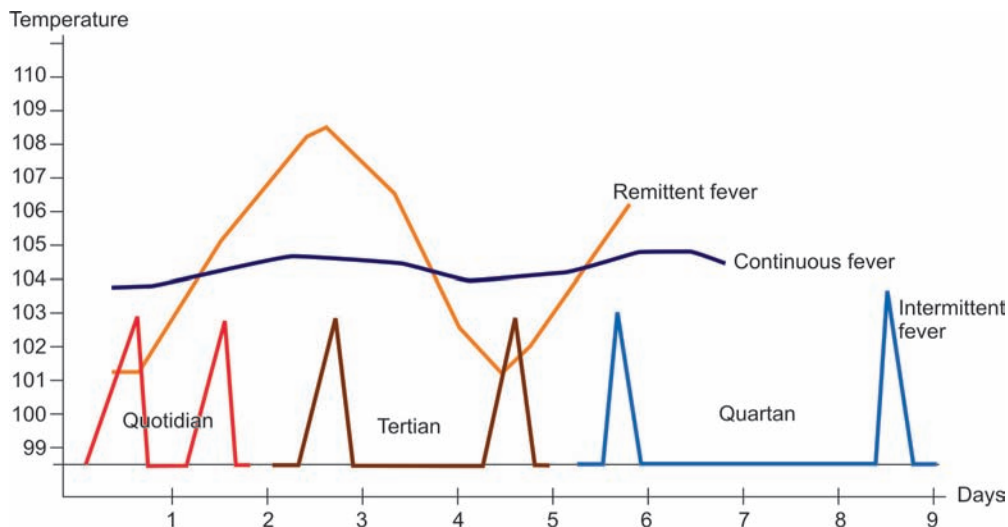


Fig. 1.17: Different types of fever.

haemorrhage. Discolouration can occur after coloured food intake, tobacco chewing, Addison's disease, iron tablets intake. Central cyanosis is observed in tongue. Tongue is moist normally; dry tongue suggests dehydration, shock. Dry brown tongue is a feature of uraemia, intestinal obstruction. Mouth dryness is graded as normal (0); mild (1); moderate (2). Furring of tongue is seen in smokers, stomatitis, and poor oral hygiene. Black hairy tongue is seen in fungal infection. Bald tongue is due to atrophy of papillae. It is seen in iron deficiency anaemia, vitamin B₁₂ deficiency. Curdy coating is seen in candidiasis infection. Leukoplakia as a whitish opaque thickened epithelium may be seen; it is often associated with superficial glossitis. Congenital fissuring can occur with irregular folds. Fissuring may also be a presentation of carcinoma of tongue. Lozenge shaped loss of papillae and fissuring is seen in midline in front of the foramen caecum. Lingual thyroid may be seen posteriorly in midline. Inability to protrude tongue is seen in *ankyloglossia* in tongue tie, advanced carcinoma tongue infiltrating the genioglossus muscle. While protruding tongue may deviate towards same side in hypoglossal nerve palsy.

Hiccup

It is spasmodic contraction of diaphragm. It is commonly idiopathic which subsides on its own. Post-

operative hiccup is common. It is due to increased abdominal pressure, pushing the diaphragm upwards. There may be paralytic ileus, gastric dilatation, and intestinal obstruction. Peritonitis involving diaphragmatic surface can cause hiccup. Renal failure causes hiccup.

Crepitus

It is crackling or grating sensation felt on palpation of subcutaneous tissue or joint or bone. Crackling sensation is felt when air is under the palpating fingers. Pockets of air moves in between separated subcutaneous or soft tissues causing crackling feel. Grating sensation is felt in bone or joint as crepitus.

Types

Crepitus in subcutaneous (surgical) emphysema: It is crackling sensation felt with gentle pressure under examining fingers similar to a palpating horse hair mattress. It can often be heard by placing a stethoscope over the surface. Subcutaneous emphysema is better felt (often seen as bull neck) in neck, shoulder and chest wall. Causes of subcutaneous emphysema are—traumatic (Fracture ribs, laryngeal injury, tracheostomy, fracture skull with air sinus like frontal sinus injury); after surgery air may get trapped in the subcutaneous plane prior to closure of skin, after

laparoscopic surgery; infective (in gas gangrene); after oesophageal rupture (Boerhaave's syndrome—here mediastinal emphysema, subcutaneous emphysema, shock, toxicity occurs).

Crepitus of tenosynovitis: It is seen in de Quervain's tenosynovitis. Here hand is laid upon arm above the wrist, and the patient is asked to close and open the hand. Crepitus is felt at the junction of extensor pollicis brevis and abductor pollicis longus crossing the extensor carpi radialis longus and brevis. **Crepitus of bursitis** is felt when lining is rough or contains loose fibrinous particles.

Joint crepitus: It is felt when affected joint passively moved by one hand, and by placing other hand over the suspected joint. It can be—*fine, even* crepitations of chronic and subacute joint diseases; coarse, irregular crepitations of osteoarthritis; *a click* due to loose body or displaced cartilage. **Bone crepitus** is elicited over the fracture segments of the bone when two fragments are moved against each other. A grating sensation is typical. But this should be elicited with utmost gentleness; only when radiological doubt exists. Crepitus is an unmistakable, diagnostic sign of fracture.

Skin Changes and Eruptions (Figs 1.18 to 1.22)

Macule: It is not raised above the skin; there is alteration in colour of skin; it is seen but not felt; capillary naevi or erythema blanch on pressure, purpuric macules do not blanch on pressure. Macules can be generalised; as seen in typhoid, syphilis, purpura; localised type is called as *roseolar*.

Papule: It is raised tiny nodule; usually of few mm in size; it may be epidermal or dermal; seen in measles, chickenpox, smallpox, drugs like sulfonamides, occasionally in tuberculosis, sarcoidosis.

Vesicles: They are small blisters; elevations from epidermis containing clear or milk like fluid within; seen in chickenpox, smallpox, herpes.

Pustules: They are epidermal elevations containing pus; due to bacterial like streptococcal infection. **Granule** is projection of < 2 cm in size. **Nodule** is large usually solid projection from the skin of more than 2 cm in size.



Fig. 1.18: Drug induced allergic rashes on the back extensively involved.



Fig. 1.19: Alopecia scalp developed after chemotherapy.

Wheal: It is elevated patch on the skin with centre paler than the periphery; it is oedematous elevation with itching; seen in allergical conditions.

Café au lait spots: They are coffee brown coloured patches in the skin; more than 5 in number with each



Fig. 1.20: Herpes zoster infection.



Figs 1.21A and B: Skin rashes and skin vesicles in two different patients.



Fig. 1.22: Radiation dermatitis both sides of neck and face.

more than 1.5 cm in size are significant; seen in von Recklinghausen's disease of neurofibromatosis with regular outline and deep indentations; occasionally also seen in Albright's syndrome as irregular outline.

Petechiae: Tiny haemorrhagic spots less than 1 mm in size.

Purpura: Haemorrhagic spots of 2-5 mm in size.

Ecchymosis: Haemorrhagic spots more than 5 mm in size.

Haematoma: Haemorrhage causing elevation of skin.

Dry skin: Seen in dehydration and myxoedema.

Moist skin: Seen in myocardial infarction, shock of sudden onset (haemorrhage), toxic thyroid.

Thick skin: Seen in myxoedema, acromegaly, and scleroderma.

Thin skin: Seen in old people, and wasting diseases.

Pinched skin a feature of dehydration, malnutrition.

Falling of hair: Seen in infectious fevers like typhoid, chemotherapy for malignancies, drugs and hereditary. Patchy hair loss is seen in alopecia areolata, syphilis. Loss of hair in outer third of eyebrow: Seen in leprosy, myxoedema. Absence of axillary, pubic and facial hairs is seen in hypopituitarism, hypogonadism.

Excessive hair growth in women is seen in Cushing's syndrome, adrenocortical syndrome.

General examination is done for proper diagnosis and differential diagnosis; for selecting the patient for anaesthesia; to decide type of surgery to be done (mesh hernioplasty is done in inguinal hernia if patient is having chronic respiratory disease or if there is poor abdominal muscle tone); to predict the prognosis (patients with gastrointestinal cancer showing palpable supraclavicular lymph node means poor prognosis; patient with carcinoma breast having spread to bones/lungs carry poor prognosis).

Other General Examinations

Other general examinations to be done are:

Head and neck region: Cranial nerve functions; eyes (visual field, pupils for equality and reaction, accommodation reflex, conjunctiva, eyeball movements,

fundus examination); mouth and pharynx (teeth, gums, soft palate movement, tongue, tonsils, lip); neck (movements of neck, neck veins, neck nodes, carotid pulse, trachea, thyroid).

Upper limbs: General look of hands, forearm, arm (wasting); vascular system; nervous system (sensation, muscle power, muscle tone, reflexes); axillary nodes; joints and movements; fingers and nails.

Thorax: Chest look; dilated veins; swelling (**Fig. 1.23**); pulsations; breasts; apex beat; lungs and heart.

Abdomen: Abdominal wall (umbilicus, scar, dilated veins); reflexes of abdomen; visible peristalsis; visible pulsation; hernial orifices; palpation; percussion; auscultation; rectal digital examination; per vaginal examination if needed in females; examination after catheterization if needed.

Lower limbs: Examination of feet, legs, thighs; feeling of peripheral pulses; nervous system in the lower limbs; oedema feet; varicose veins; examination in standing position; joints; inguinal nodes.

Examination of genitalia: Testis (its size, texture, presence of hydrocele); epididymis; vas deferens, skin over the scrotum; penis for phimosis, balanoposthitis, chordee, hypospadias.

Skeletal system: spine and skull.



Fig. 1.23: Systemic examination is a must. Note the chest wall swelling in this patient. Clothings should be removed properly while examining the patient. This swelling may be secondary in the rib or primary tumour.

Examination of Faeces

It gives indirect evidence of different pathologies in the gastrointestinal tract. The quantity—copious/scanty; consistency—liquid/semisolid/semiformed/formed/hard; colour—black—in upper GI bleed, iron or bismuth intake/pale coloured stool is seen in obstructive jaundice (absence of bile in the bowel), rapid transit of stool in diarrhoea, malabsorption, chronic pancreatitis; odour—offensive in jaundice, semen like odour in acute amebic dysentery, odourless in acute bacillary dysentery; type—slimy stool in carcinoma colon, colitis of different causes; purulent stool in bacterial dysentery; blood in stool in different conditions. **Melaena**—is black, tarry, foul smelling stool seen in upper GI bleed (**Fig. 1.24**); red pigmented clots (Maroon coloured) in Meckel's diverticulum; red currant jelly in intussusception; bright red coloured in rectal and anal diseases. **Steatorrhoea** is large quantity, pale, porridge-like stool that, sticks to lavatory and is difficult to flush. It is due to severe degree of pancreatic insufficiency causing malodorous, voluminous stool which floats on the water. Patient passes quantity of fat that separates from the non-fatty part of the faecal matter that resembles melted butter that become solid again. **Pipe stem stool** occurs in rectal stenosis usually due to malignant rectal stricture. **Toothpaste stool** is seen in Hirschsprung's disease. Spurious diarrhea is seen in carcinoma colon.

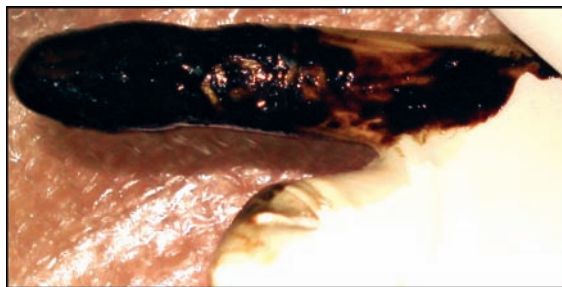


Fig. 1.24: Typical black, foul smelling tarry coloured stool—seen in upper gastrointestinal bleeding.

ECOG performance status (Eastern Cooperative Oncology Group): This performance status is used as a guide to plan the therapy. Other scale used is Karnofsky scale.

ECOG (Zubrod) scale	Performance	Karnofsky score
0	Fully active and able to carry out work without restriction	100%
1	Symptoms restrict strenuous physical activity but ambulatory and able to carry light sedentary work	80-90%
2	Ambulatory but unable to carry out work; up and about > 50% waking hours	60-70%
3	Only limited self care; confined to bed or chair for more than 50% of waking hours	40-50%
4	Completely disabled; confined to bed or chair	20-30%

Local Examination

Inspection

It is observing the diseased area carefully for clinical features. It should be done with proper complete exposure of the part; compared with normal side.

Palpation

It is done by feeling of affected part using hand and fingers.

Percussion

It is tapping of the affected area directly using flexed finger (direct method) or using pleximeter finger and

percussion finger (indirect method). Percussion is used over sternum, abdomen (ascites, over mass to find out note, liver dullness), respiratory system (in pleural effusion, pneumothorax).

Auscultation

Stethoscope is used to hear heart sounds, abnormal sounds like adventitious breath sounds, altered bowel or absence bowel sounds; bruit over vessel or organ.

Examination of regional lymph nodes is essential.

Movements and measurements are also often important.

Note:

Sequence of events needed in approaching all patients are as follows –

1. Detailed complete proper history.
2. Complete clinical examination.
3. Clinical analysis, diagnosis and differential diagnosis.
4. Evaluation of the patient routine/general and specific in relation to the patient's probable diagnosis.
5. Final diagnosis.
6. Planning the therapy/treatment which are best suitable for the patient.
7. Implementing the treatment surgical/conservative (like drugs)/radiotherapy, etc.
8. Follow up of the patient.

2

Examination of an Ulcer

Definition

An ulcer is a break in the continuity of the covering epithelium, either skin or mucous membrane due to molecular death.

Parts of an Ulcer (Fig. 2.1)

1. **Margin:** It may be regular or irregular. It may be rounded or oval in shape.
2. **Edge:** Edge is the one which connects floor of the ulcer to the margin.

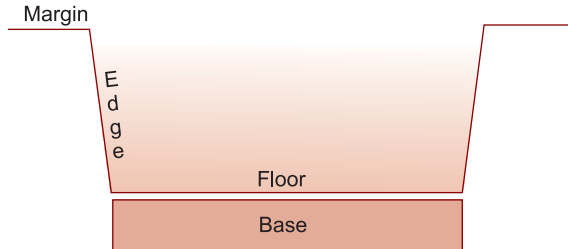


Fig. 2.1: Parts of an ulcer.

Different edges are (Fig. 2.2):

Sloping edge: It is seen in healing ulcer. Its inner part is red because of red, healthy granulation tissue. Its middle part is white due to scar/fibrous tissue. Its outer part is blue due to epithelial proliferation.

Undermined edge is seen in tuberculous ulcer. Disease process advances in deeper plane (in subcutaneous tissue) whereas epidermis (skin) proliferates inwards.

Punched out edge is seen in gummatous (syphilitic) ulcer and trophic ulcer. It is due to end arteritis.

Raised and beaded edge (pearly white) is seen in rodent ulcer (BCC). Beads are due to proliferating active cells.

Everted edge (rolled out edge): It is seen in carcinomatous ulcer due to spill of the proliferating malignant tissues over the normal skin.

3. **Floor:** It is the one which is seen. Floor may contain discharge, granulation tissue.

4. **Base:** Base is the one where ulcer rests. It may be bone or soft tissues.

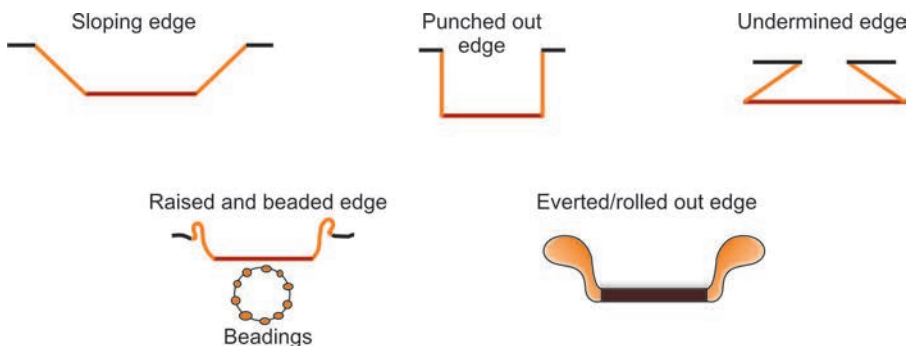


Fig. 2.2: Types of edges in different ulcers.

Classifications

Classification I (Clinical)

1. *Spreading ulcer*: Here edge is inflamed and oedematous.
2. *Healing ulcer*: Edge is sloping with healthy pink/red granulation tissue with serous discharge.
3. *Callous ulcer*: Floor contains pale unhealthy granulation tissue with indurated edge/ base. *Ulcer has no tendency to heal*. It lasts for many months to years (**Fig. 2.3**).



Fig. 2.3: Callous ulcer without any sign of healing and, without any granulation tissue. It is due to callous attitude of the patient.

Classification II (Pathological)

1. *Specific ulcers*:
 - Tuberculous ulcer.
 - Syphilitic ulcer: It is punched out, deep, with 'wash-leather' slough in the floor and indurated base.
 - Actinomycosis.
 - Meleney's ulcer.
2. *Malignant ulcers*:
 - Carcinomatous ulcer.
 - Rodent ulcer.
 - Melanotic ulcer.

3. Non-specific ulcers:

- *Traumatic ulcer*: It may be mechanical (dental ulcers in the tongue), physical (electrical burn), and chemical (alkali injury).
- *Arterial ulcer*: Atherosclerosis, TAO.
- *Venous ulcer*: Gravitational ulcer, post-phlebotic ulcer.
- *Trophic ulcer*: Bed sore; perforating ulcers in the sole.
- *Infective ulcers*: Pyogenic ulcer.
- *Tropical ulcers*: It occurs in tropical countries. It is callous type of ulcer, e.g. Vincent's ulcer.
- *Ulcers due to chilblains and frostbite (cryopathic ulcer)*.
- *Martorell's hypertensive ulcer*.
- *Bazin's ulcer*.
- *Diabetic ulcer*.
- *Ulcers due to leukaemia, polycythaemia, jaundice, collagen diseases, lymphoedema*.
- *Cortisol ulcers* are due to long time application of cortisol (steroid) creams to certain skin diseases. These ulcers are callous ulcers, last for long time and require excision and skin grafting (**Fig. 2.4**).



Fig. 2.4: Non-healing ulcer with pale unhealthy granulation tissue.

Classification III (Wagner's Grading)

Grade 0—preulcerative lesion/healed ulcer.

Grade 1—superficial ulcer.

Grade 2—ulcer deeper to subcutaneous tissue exposing soft tissues or bone.

Grade 3—abscess formation underneath/osteomyelitis.

Grade 4—gangrene of part of the tissues/limb/foot.

Grade 5—gangrene of entire one area/foot.

Different discharges in an ulcer (as well as from a sinus)

Serous: in healing ulcer

Purulent: in infected ulcer

Staphylococci: yellowish and creamy

Streptococci: bloody and opalescent

Pseudomonas: greenish colour

Bloody: malignant ulcer, healing ulcer from healthy granulation tissue (**Figs 2.5A and B**)

Seropurulent

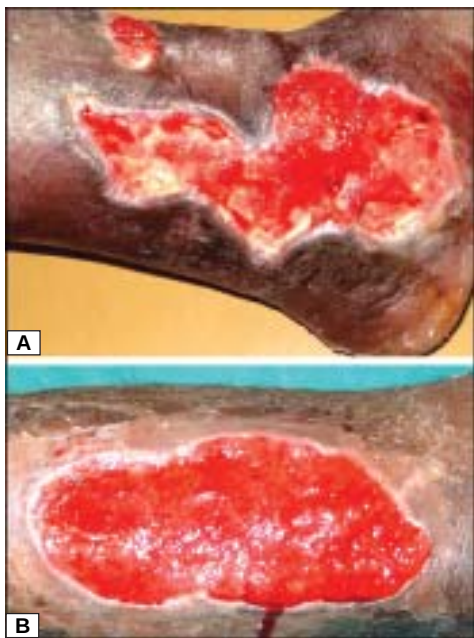
Serosanguineous: serous and blood

Serous with sulphur granules: Actinomycosis

Yellowish: Tuberculous ulcer

Causes of Ulcer Becoming Chronic in the Skin

Causes of ulcer becoming chronic in the skin are: Recurrent infection; trauma; absence of rest; poor blood supply; hypoxia; oedema of area; loss of sensation; malignancy; specific cause like tuberculosis, fibrosis, periostitis or osteomyelitis of the underlying bone.



Figs 2.5A and B: Ulcers with healthy granulation tissue. Ulcer is ready for split skin grafting.

History

Name:

Sex:

Age: Certain diseases or ulcers may be more common in certain age groups.

Occupation: Venous ulcers are more common in individuals whose occupation requires long hours of standing like nurses, surgeons, traffic policemen, watchmen and bus conductors.

Place:

Chief complaints: History of ulcer and its duration should be mentioned. History of specific condition related also should be mentioned.

History of Present Illness

Mode of Onset and Progression

It is the initial way of formation of an ulcer. It may be after an attack of cellulitis of the part which causes skin necrosis and later sloughs off, or after trauma which breaks the continuity of the epithelium or spontaneously due to any cause. Traumatic ulcer may heal fast or may progress into chronicity if it is on a joint or due to improper rest to the part or if patient is diabetic or due to other precipitating causes. Common cause of ulcer is trauma. Even minor trauma can cause extensive necrotising fasciitis and ulcer later. Often patient will be having the idea of the cause of ulcer. Tuberculous lymphadenitis leading into collar stud abscess eventually may form a fistula or an ulcer. Ulcerative lesion may be rodent ulcer, carcinomatous ulcer or melanotic ulcer originating spontaneously. Syphilis may lead into gummatous ulcer. Ulcer may occur as a result of varicose veins due to chronic venous hypertension or arterial insufficiency as in ischemic ulcer or over pre-existing scars like of burn scar. Regressing or progressing of an ulcer formed in specific method is important. Ulcer may often heal spontaneously and reform later repeatedly in the same site, e.g. Formation of an ulcer in a pre-existing burn scar or formation of venous ulcer repetitively around ankle. Here ulcer heals by rest and reforms by trauma or other precipitating causes. If it is progressing then method of progressing is also noted. Change in size, shape, depth, discharge during progression period should be asked (**Fig. 2.6**).



Fig. 2.6: Ulcer over lateral margin of the tongue due to dental injury. It is an acute ulcer.

Duration

Ulcer is of long duration like in chronic venous ulcer or of short duration like in acute ulcer after trauma or cellulitis.

Pain

Ulcer may be painful or painless. Often ulcer is painless to begin with but may eventually become painful like malignant ulcers due to secondary bacterial infection or infiltration to deeper plane or nerve ending. Some ulcers are painful to begin like acute ulcer, but becomes painless once it turns to chronicity. Its time of onset, progress, and severity should be asked. Trophic ulcers, syphilitic ulcer, ulcers of neurological diseases like spinal injury/spinal diseases/peripheral neuropathy/tabs dorsalis are painless. Pain may interfere with patient's daily routine activities like walking, eating, bathing, defecation, etc. Tuberculous ulcer is usually painful.

History of Fever

Presence of fever signifies existing acute inflammation in an ulcer or in surrounding area.

Discharge from Ulcer

Whether ulcer is having discharge or not is significant. Discharge can be assessed by looking at dressing pads also. Discharge may be profuse, scanty or absent. Patient often gives history of the quantity of dressing pads soaked and its colour. Colour and

smell of discharge is important to be considered, whether discharge is serous or purulent and often type of bacterial infection involved like in pseudomonas infection the discharge is greenish in colour.

Number of Ulcers

Often ulcers can be multiple (**Fig. 2.7**). If it is so, which ulcer developed first and which one later should be asked.



Fig. 2.7: Multiple ulcers all over the body in a malnourished immunosuppressed individual.

Associated Symptoms

History of presence of varicose veins; claudication, rest pain of arterial insufficiency should be asked for.

Past History

Past history of ulcer treated by dressing/drugs/skin grafting/hospital stay should be asked in detail. Number of days hospitalized, time taken up for healing of the ulcer should be noted. Previous history of treatment for tuberculosis, syphilis, diabetes or any other illness is important.

History suggestive of associated disease/treatment history like for tuberculosis, tabs dorsalis, spinal diseases or diabetes mellitus has to be asked. If patient

is on treatment for any of such ailment, type of drugs taken, dose and method of intake should be asked.

Personal History

History of alcohol consumption/smoking/tobacco chewing/history of sexual contact/dietary habits are also important. Duration of such habits, quantity are also important. It has got direct relation to ulcer formation or ulcer healing or treatment strategy. *Altered appetite or weight loss* can also be mentioned under personal history—may be due to advanced malignancy or tuberculosis.

Family History

Family history of any specific disease should be asked.

General Examination

Doing a detailed general examination is very essential. Presence of anaemia/oedema/jaundice/clubbing/lymphadenopathy/raise in temperature/attitude of the patient/nutritional assessment by skin texture, subcutaneous fat, weight, body mass index/any other relevant findings should be mentioned. Rate and volume of radial pulse/palpation of all peripheral pulses/blood pressure should be noted. Malignant tumor infiltrating nerves or ulcer with a chronic scar or large chronic ulcer or painful acute ulcer can alter the attitude of the limb. Increased pulse rate and temperature suggests ulcer with acute inflammation. Features suggestive of tuberculosis, vascular disease, spinal disease, syphilis or neurological diseases should be looked for.

Local Examination of an Ulcer

Inspection (Figs 2.8A to C)

Site of an Ulcer

Exact anatomical location of the ulcer is noted. It is mentioned in relation to particular anatomical point usually bony point. Venous ulcers occur over malleoli around ankle (**Fig. 2.9**). Basal cell carcinomatous ulcer/rodent ulcer occurs in the face commonly above the line joining angle of the mouth to the ear lobule (common site is inner canthus of eye) (**Fig. 2.10**).



Figs 2.8A to C: Inspection of ulcer for its site, size, shape, margin, edge, floor and surrounding area.



Fig. 2.9: Inspect the limb in standing position for evidence of varicose veins in case of suspected venous ulcer.

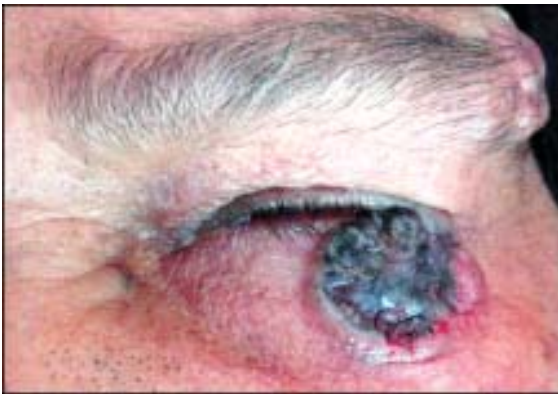


Fig. 2.10: BCC (rodent ulcer) which is pigmented involving lower eyelid—a common site.

Tuberculous ulcer occurs commonly in the neck, axilla, chest wall or groin. It can occur anywhere in the body. Syphilitic gummatous ulcer occurs over subcutaneous bones like tibia, sternum, palate or skull. Trophic ulcer occurs in the sole of the foot (heel, over the heads of the metatarsals). Malignant ulcers can occur anywhere in the skin. Meleney's ulcer occurs over the perineum, groin and lower abdominal wall.

Size of an Ulcer

Ulcer size should be measured both vertically and horizontally using a measuring tape. Tape may be placed over sterile gauze covering the ulcer to measure (**Fig. 2.11**).



Fig. 2.11: Ulcer should be measured both vertically and horizontally.

Shape of an Ulcer

Ulcers of different causes may have different shapes. Venous ulcer is vertically *oval* in shape. Tuberculous ulcer is *circular* in shape. Malignant ulcer is *irregular* in shape. *Serpiginous* ulcer looks like a serpent. Here healing occurs in one place; while disease extends in another/adjacent place.

Number

Malignant ulcer is usually solitary. Venous ulcers can be multiple. Tuberculous ulcers can be multiple.

Margin of an Ulcer

Margin is whether regular/irregular/well-defined/ill-defined should be observed. Margin is the junction of the normal skin around to the outermost end of the edge.

Edge of an Ulcer

Edge is from the floor to margin. Different edges occur in different conditions.

Sloping edge occurs in healing ulcer. It shows three zones from inside out. First is red zone due to central

healthy red granulation tissue; second is middle blue zone consisting of active growing epithelium; third is outermost white zone consisting of fibrous tissue and scar. It is seen in *healing ulcer* (**Fig. 2.12**).



Fig. 2.12: Healing ulcer with healthy granulation tissue. Note the sloping edge.

Punched out edge is edge that run deeply perpendicular to the skin margin. It is commonly seen in *trophic ulcer* (due to localized deep inflammation) and *gummatous ulcer* (due to end arteritis obliterans). Disease is localized to ulcer area and does not spread to surrounding structures (**Fig. 2.13**).



Fig. 2.13: Trophic ulcer heel—typical punched out edge.

Undermined edge is one where edge is burrowed deep and lateral to the skin margin. *Edge in tuberculous ulcer* is typically undermined. It is due to faster spread of tuberculosis in subcutaneous plane than skin. Overlying skin which is pointing towards the center of the ulcer is bluish, thin, and friable (**Figs 2.14A and B**).

Raised and beaded edge is seen in *rodent ulcer* (BCC ulcer). Beads are pearly white in colour and are due to actively multiplying malignant cells. Probably in between these beads are predominantly the dormant inactive cells (**Fig. 2.15**).

Everted edge/rolled out edge is one which fills, heaps and spills outward from the edge towards margin. It is typical of *epitheliomatous ulcer* (squamous cell carcinoma/malignant ulcer). It signifies rapidly growing tissues (**Fig. 2.16**). Ulcer showing proliferation (usually in the edge) is proliferative ulcer. Growth showing ulceration is ulcerative growth.

Spreading ulcer shows oedematous, inflamed edge. *Aphorism*—syphilis bites; tuberculosis nibbles.



Fig. 2.14: Tuberculous ulcer foot with undermined edge.



Fig. 2.15: Basal cell carcinomatous ulcer (rodent ulcer) with beaded edge.

Floor of the Ulcer

Floor is the one what is seen. It rests on the base. (Base is not seen; it is only felt). Floor may contain red granulation tissue in healing ulcer; pale, unhealthy granulation tissue in non-healing ulcer; thick slough without any granulation tissue in callous chronic ulcer; pigmented tissue in melanoma or pigmented BCC or pigmented SCC (rare). Wash leather slough/wet chamois leather slough is seen in gummatous ulcer. Wash leather slough often also seen in post-irradiated necrotic slough in the floor of malignant ulcer. Often moving maggots may be present in the floor. They eat necrotic dead tissue only. Cultivated maggots are used as therapeutic desloughing agent. *Slough* is dead soft tissue *in situ* (**Figs 2.17 to 2.19**).



Fig. 2.16: Squamous cell carcinoma scalp. Note the ulceroproliferative lesion with everted edge.

Discharge from Ulcer Bed

It can be serous (healing ulcer), serosanguinous, bloody (malignant ulcer), purulent (infective ulcer); colour of discharge has to be noted—greenish in pseudomonas infection (**Fig. 2.20**). *Quantity, quality, colour and smell* of discharge should be assessed. Dried up discharge looks like *scab* in the ulcer floor.



Fig. 2.17: Ulcer leg with exposed bone. Patient underwent local rotation flap to cover. Area from where the flap is rotated is covered with split skin graft. Skin grafting is not possible over the exposed bone.



Fig. 2.18: Ulcer in the foot initially with slough, and later after slough excision and regular dressings. Area requires skin grafting later. *Slough* is dead soft tissue *in situ*.

Surrounding area has to be examined for inflammation, oedema, eczema, scars. Inspection of the entire part/limb should be done for deep vein thrombosis, arterial disease, and neurological causes.

Always inspect opposite limb also.

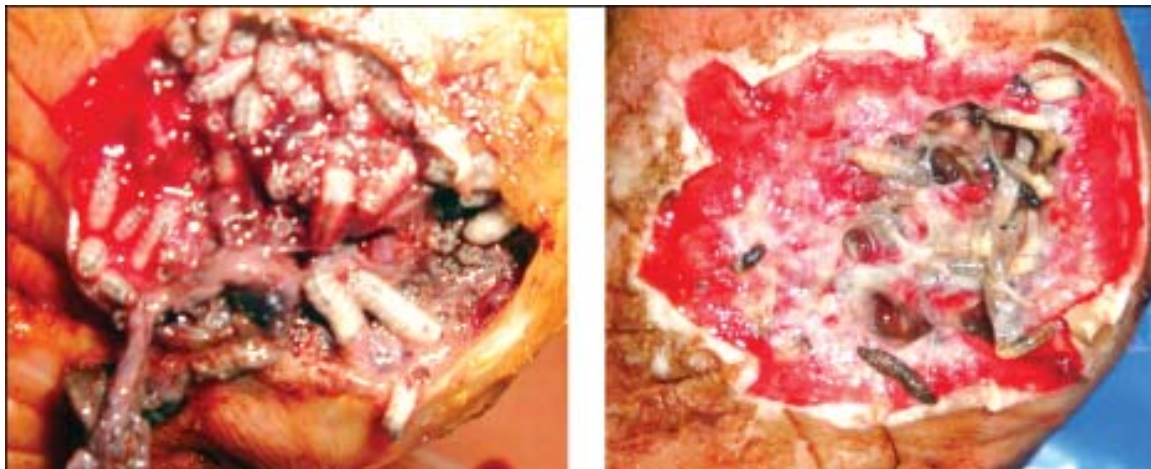


Fig. 2.19: Maggots in an ulcer.



Fig. 2.20: Non-healing ulcer foot in a diabetic patient with pseudomonas infection. Note the greenish discharge in the wound. Pseudomonas infection is commonly hospital acquired.

Palpation

Tenderness should be elicited over the edge, base and surrounding area. Acute ulcers are tender. Chronic ulcer is usually non-tender but can be tender if there is secondary infection, involvement of deeper structures like periostitis in venous ulcer. Malignant ulcer is non-tender to begin with. It may only become tender in later period when it infiltrates into deeper plane (**Figs 2.21A and B**).

Warmness over *surrounding area* signifies acute inflammation.

Palpation of Edge for Tenderness and Induration

Induration is feeling of hardness. It often suggests carcinoma. In chronic ulcer hardness can be felt because of thick fibrosis (**Fig. 2.22**).



Figs. 2.21A and B: Checking the temperature in surrounding area and comparing opposite/normal area.



Fig. 2.22: Palpating the edge for tenderness and induration.

Palpation of Base for Induration/Fixity

Base is the one on which ulcer lies. Base may be fascia, soft tissues or bone. If base is formed by bone then ulcer is fixed and non-mobile. Mobility should be checked in two planes. Induration of base is important in carcinoma. Hunterian chancre also shows induration.

Depth of Ulcer

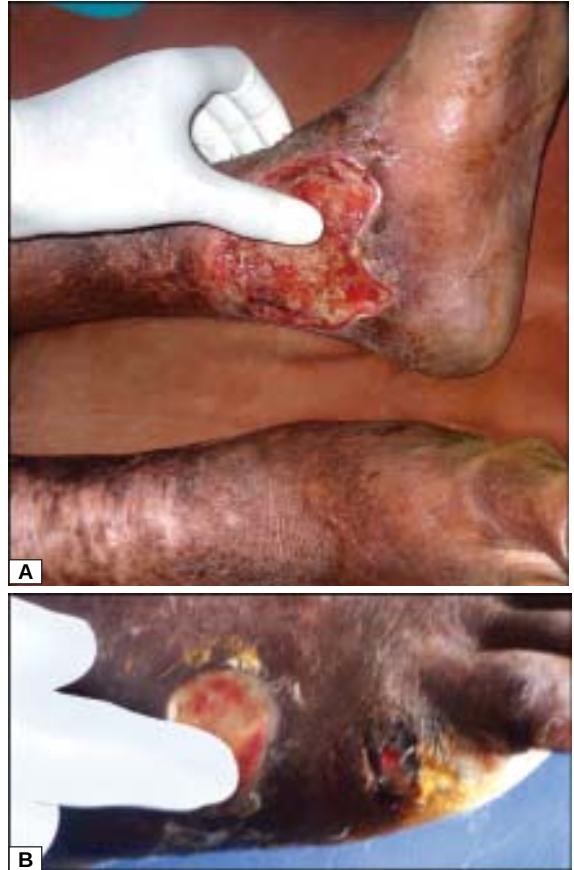
Trophic ulcer is deep with bone as its base. Depth is measured in mm.

Bleeding on Palpation and Touching

Floor and edge should be palpated for this sign after wearing a sterile glove. Malignant ulcer is vascular and friable hence bleeds on touch. Healthy and exuberant granulation tissues in the floor can bleed on touch (**Figs 2.23A and B**).

Palpation of Deeper Structures and its Relation to Ulcer

Bone and soft tissues should be palpated. Bone thickening signifies periostitis or osteomyelitis due to ulcer penetration. It is felt by running thumb firmly over the surface of the bone. It is commonly elicited in lower tibia and malleoli in case of venous ulcer; in calcaneum in trophic ulcer. Mobility also of an ulcer should be checked by wearing a glove. Ulcer is held firmly at two opposite points over the margin and tried



Figs 2.23A and B: Palpation may cause bleeding on touch in healthy granulation tissue or carcinoma. Base of an ulcer also should be palpated for tenderness and induration.

to move over the base. It should be checked in two perpendicular directions (**Figs 2.24A to 2.25B**).

Surrounding skin should be looked for oedema, inflammation, pigmentation, pallor (**Fig. 2.26**).

Inspection

Examination of Adjacent Joint

Joints are examined for both active and passive movements. Active movements are done by the patient. Passive movements are elicited by the clinician (**Figs 2.27A and B**).

Examination of Regional Lymph Nodes

Examination of regional lymph nodes is essential — *Tender*, palpable regional lymph nodes are found in



Figs 2.24A and B: Bone thickening should be felt by palpation over proximal and distal part of the ulcer. Here ulcer is in ankle region and so thickening of tibia and calcaneum should be checked.

acute infective conditions. *Shotty, firm, discrete* lymph nodes are felt in Hunterian chancre. Lymph nodes are not enlarged in BCC/rodent ulcer as malignant cells block the lymphatics early. *Stony hard*, initially discrete and mobile lymph nodes, but later when advanced fixed to deeper structures are features of secondaries from carcinoma. Initially regional lymph nodes may get enlarged due to infection as such, and not due to primary existing carcinoma. Such nodes are usually firm, not hard and may regress by trial antibiotic therapy. Lymph nodes enlarged due to sepsis may get suppurated and may form an abscess as *soft, tender swelling*. Lymph nodes involved by tuberculosis are *matted, firm*, often may lead to cold abscess or collar stud abscess.

Examination of Arterial Pulse

Examination of arterial pulse peripherally in relation to ulcer should be done (**Figs 2.28A and B**).



Figs. 2.25A and B: Mobility of an ulcer should be checked. If there is free mobility it means it is not fixed to bone. If mobility is absent then it could be fixed to bone.



Fig. 2.26: Surrounding area should be palpated for relevant findings.



Figs 2.27A and B: Joint proximal to the ulcer area should be checked for any change in movement. Fibrous ankylosis and total loss of joint movement can occur. Ankle joint should be examined by holding lower leg flexed with left hand and right hand placed just distal to ankle joint (with heel off the ground) to check for dorsiflexion (normal is 25°) and plantar flexion (normal 35°). Inversion (20°) and eversion (20°) is checked by holding the calcaneum with one hand and foot distally with other hand.

Examination for Varicose Veins

Varicose veins are examined in standing position and all relevant tests should be done in case of venous ulcers.

Examination of Peripheral Pulses

Examination of peripheral pulses should be done to confirm if there is any ischaemia (**Fig. 2.29**).

Examination of Spine and Neurological System

Examination of spine and neurological system like sensation and muscle power in the region and specific segments (**Fig. 2.30**).

Gait of the Patient

Gait of the patient should be checked to find out the severity of loss of function due to ulcer (**Fig. 2.31**).



Figs 2.28A and B: Regional lymph nodes should be palpated for enlargement. In lower limb ulcer, vertical superficial group of inguinal nodes are palpated. External iliac nodes are also checked above and on medial aspect of the inguinal ligament. Its enlargement signifies severity of the disease.



Fig. 2.29: Palpation of peripheral pulses to find out ischemia.

Systemic Examinations

Systemic examinations like of abdomen, respiratory and cardiovascular system should be done properly (**Figs 2.32A and B**).

Induration of an Ulcer

Induration is a clinical palpatory sign which means a specific type of hardness seen in the diseased tissue. It is obvious in well differentiated carcinomas (squamous cell or adenocarcinomas). It is better felt in



Fig. 2.30: Examination of neurological system is a must. Touch sensation and sense of positions are commonly tested. Checking the muscle power and grading should also be done. Spine should be examined for tenderness/deformity.



Fig. 2.31: Checking the gait in an ulcer patient.

squamous cell carcinoma. It is also observed in long-standing ulcer with fibrosis. It is absent or less in poorly differentiated carcinomas and malignant melanoma. Less indurated carcinoma is more aggressive. Specific types of indurations are observed in venous diseases and chronic deep venous thrombosis. *Brawny* induration is a feature of an abscess. Induration is felt at edge, base and surrounding area of an ulcer. Induration at surrounding area signifies extent of disease (tumor). Outermost part of the indurated area is taken as the point from where clearance for wide excision is planned. Hunterian chancre shows induration.

Granulation Tissue

It is seen on the floor of an ulcer consisting of proliferating new capillaries and fibroblasts intermingled with RBCs and WBCs with thin fibrin cover over it. It contains fine capillary loops, fibroblasts with thin fibrin and plasma covering.



Figs 2.32A and B: Systemic examination like of abdomen, respiratory, cardiovascular system, spine, neurological examination is a must.

Types:

Healthy granulation tissue: It is seen in a healing ulcer. It has got sloping edge. It bleeds on touch. It has got serous discharge. Skin grafting takes up well in an ulcer with healthy granulation tissue (**Fig. 2.33**). Streptococci growth in culture should be less than 10^5 /gram of tissue before skin grafting.

Unhealthy granulation tissue: It is pale with purulent discharge. Its floor is covered with slough. Its edge is inflamed and oedematous. It is seen in spreading ulcer.

Unhealthy, pale, flat granulation tissue: It is seen in chronic nonhealing ulcer (*callous ulcer*).



Fig. 2.33: Ulcer with healthy granulation tissue ready for split skin grafting.

Exuberant granulation tissue (*Proud flesh*): It occurs in a sinus wherein granulation tissue protrudes out of the sinus orifice like a proliferating mass. It is commonly associated with a retained foreign body in the sinus cavity (**Fig. 2.34**).

Pyogenic granuloma: It is a type of exuberant granulation tissue. Here granulation tissue protrudes out from an infected wound or ulcer bed presenting as well localised, red swelling which bleeds on touching.



Fig. 2.34: Exuberant granulation (*proud flesh*) in an ulcer. It should be scooped out using Volkman's scoop prior to skin grafting.

Investigations for an Ulcer

Study of discharge: Culture and sensitivity, AFB study, cytology.

Edge biopsy: Biopsy is taken from edge because edge contains multiplying cells. Usually two biopsies are taken. Because of central necrosis, biopsy may be inadequate if taken from the centre. But in recurrent post radiation malignant ulcer biopsy is taken from centre, as active proliferating cells are present in the centre not in periphery due to vascular fibrosis in the edge by radiotherapy. **X-ray of the part** to see periostitis or osteomyelitis (**Fig. 2.35**).

FNAC of the regional lymph node. Other tests like Chest X-ray, Mantoux test in suspected case of tuberculous ulcer. Haemoglobin and albumin levels in blood are important. Granulation tissue will not develop if Hb% is below 10 gm%; and if albumin is less than 3 gm%.

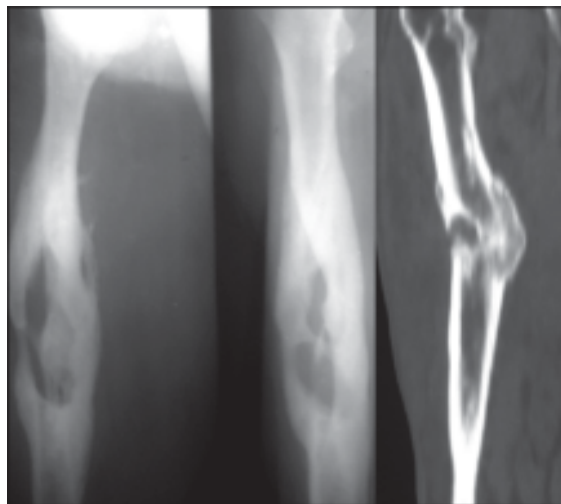


Fig. 2.35: X-ray showing osteomyelitis with sequestrum inside. Osteomyelitis prevents ulcer healing. Bone thickening on clinical examination is typical.

Assessment of an Ulcer

Cause of an ulcer should be found—diabetes/venous/arterial/infective. **Clinical type** should be assessed. **Assessment of wound** is important—anatomical site; size and depth of the wound; edge of the wound; mobility; fixity; induration; surrounding area; local blood supply (**Fig. 2.36**). **Wound perimeter** may be useful in assessing this. **Wound imaging** is done by tracing it on a transparent acetate sheet at regular intervals. **Presence of systemic features;** regional nodal status; function of the limb/part; joint movements; distal pulses; sensations should be assessed. **Severity** of infection should be assessed—culture of discharge. **Specific investigations** like edge biopsy; X-ray of part; blood sugar; arterial/venous Doppler; angiogram are done.

Trophic Ulcer

It occurs due to impaired nutrition, defective blood supply, and neurological deficit. It usually occurs in



Fig. 2.36: Ulcer after wound debridement. Adequate wound excision is done whenever there is slough and necrosis to allow granulation tissue to develop.

the heel, in relation to heads of metatarsals, buttocks, over the *ischial tuberosity*, sacrum, over the shoulder, occiput. Because there is neurological deficit trophic ulcer is called as *neurogenic ulcer/neuropathic ulcer*. Initially it begins as callosity due to repeated trauma and pressure which then suppurates and gives way through a central hole which extends into the deeper plane as *perforating ulcer (penetrating ulcer)*.

Neurological causes: Diabetic neuropathy, peripheral neuritis, tabes dorsalis, spina bifida, leprosy, spinal injury, paraplegia, peripheral nerve injury, syringomyelia. *Bedsore* is trophic ulcers.

Clinical features: It occurs in 5% of all hospitalised patients. It is painless ulcer which is punched out; nonmobile with base formed by bone (**Fig. 2.37**).

Staging of pressure sore—*Stage 1*—Non-blanching erythema—early superficial ulcer; *Stage 2*—Partial thickness skin loss—late superficial ulcer; *Stage 3*—Full thickness skin loss extending into subcutaneous tissue but not through fascia—early deep ulcer;



Fig. 2.37: Bedsore (decubitus ulcer) in sacral region—it is a trophic ulcer. It is usually with punched out edge.

Stage 4—Full thickness skin loss with fascia and underlying structures like muscle/tendon/bone, etc.—late deep ulcer (**Fig. 2.38**). *Pressure sore* is tissue necrosis and ulceration due to prolonged pressure. Blood flow to the skin stops once external pressure becomes more than 30 mm Hg (more than capillary



Fig. 2.38: Trophic ulcers in the heel in two different patients. It often requires rotation flap/transposition flap to cover.

occlusive pressure) and this causes tissue hypoxia, necrosis and ulceration. It is more prominent between bony prominence and an external surface.

Factors causing pressure sore: Normal stimulus to relieve the pressure is absent in anaesthetised patient; nutritional deficiencies worsens the necrosis; inadequate padding over the bony prominences in malnourished patients; urinary incontinence in paraplegic patient causes skin soiling—maceration—infection—necrosis.

Ulcer due to Chilblains

It is due to exposure to intense cold causing blisters, ulceration in the feet. These ulcers are **superficial**. It is due to excessive cutaneous arteriolar constriction. The condition is also called as **perniosis**.

Ulcer due to Frostbite

It is due to exposure of the part to wet cold below the freezing point. There is arteriolar spasm, denaturation of proteins and cell destruction. It leads on gangrene of the part. These ulcers here are always **deep**.

Martorelle's Ulcer

It is seen in *hypertensive* patients often with atherosclerosis. It is seen *in calf*. Often it is *bilateral*. It is *painful*. Localised necrosis of calf skin occurring suddenly with sloughing away and formation of deep, punched out ulcer extending into the deep fascia is the pathology. There is sudden obliteration of the arterioles of the calf skin. All *peripheral pulses are present*. It takes months to heal.

Bairnsdale Ulcer

It is a chronic, irregular, undermined ulcer due to *Mycobacterium ulcerans* infection. Discharge study will show acid-fast bacilli. Deep severe form with extensive dermal necrosis is called as *Buruli ulcer*.

Tropical Ulcer

It is an acute ulcerative lesion of the skin observed in tropical regions like Africa, India and South America. It is associated with lower socioeconomic group,

anaemia, and malnutrition and vitamin deficiency. It is commonly caused by *Fusobacterium fusiformis* (vincent's organisms) and *Borrelia vincenti*. There are abrasions, redness, papule and pustule formation, acute regional lymphadenitis and severe pain. Serosanguinous discharge often undermined and raised edge is common. Eventually it forms a chronic indolent large ulcer. After long time when it heals, it forms a pigmented, parchment like scar. Squamous cell carcinoma may be a occasional late complication in such disease.

Diabetic Ulcer

Causes: Increased glucose in the tissue precipitates infection; diabetic microangiopathy affects microcirculation; increased glycosylated haemoglobin decreases the oxygen dissociation; increased glycosylated tissue protein decreases the oxygen dissociation; diabetic neuropathy involves all sensory, motor and autonomous components; associated atherosclerosis affects the circulation.

Sites: Foot-plantar aspect—is the commonest site; leg; upper limb; back; scrotum; perineum, etc. Diabetic ulcer may be associated with ischaemia. Ulcer is spreading and deep (**Figs 2.39 and 2.40**).

Problems with diabetic ulcer: Neuropathy, in foot—clawing of toes, hammer toe (due to intrinsic muscle paralysis); multiple deeper abscesses; osteomyelitis of deeper bones are common; reduced leucocyte function; resistant infection; spreading cellulitis; arterial insufficiency; septicaemia; diabetic ketoacidosis; associated cardiac diseases like ischaemic heart disease.

Meleney's Ulcer (Postoperative Synergistic Gangrene, Pyoderma Gangrenosum)

It is commonly seen in postoperative wounds in abdomen and chest wall like in drainage of empyema or surgery for peritonitis. It is an acute rapidly spreading ulcer with gangrene of skin and subcutaneous tissues. It is common in old age, immunosuppressed people and when surgery is done in infected conditions. It is caused by microaerophilic streptococci and anaerobes. It begins in scrotum or perineum and rapidly spreads to groin and lower abdominal wall.



Fig. 2.39: Foot is the commonest area for diabetic infective problems. It can cause abscess, ulcer, osteomyelitis, gangrene, septicaemia. Initially patient undergoes toe amputation but later eventually may require with below knee or above knee amputation.



Fig. 2.40: Infective ulcer in the foot. Note the quantity of slough, exposed tendon and gangrenous great toe. Patient might require below knee/above knee amputation.

It can occur in other areas of skin also. Very rarely it can occur in leg or back of hand when patient is suffering from ulcerative colitis. Clinically, patient is toxic. Ulcer is rapidly spreading which is painful and

tender with large quantity of foul smelling serosanguinous discharge, showing undermined deep edge having immediate deep purple zone and outer red zone. Floor is covered with abundant unhealthy granulation tissue. Infection is severe with endarteritis of the skin leading to ulcer and destruction. It needs an emergency critical care therapy. Condition has got high mortality (**Fig. 2.41**).



Fig. 2.41: Meleney's postoperative synergistic gangrene.

Tuberculous Ulcer

It is due to breaking of the underlying cold abscess and collar stud abscess into the surface skin. It is common in neck, axilla and groin. But it can occur anywhere in the skin. Primary cutaneous tuberculosis with single or multiple ulcers also can occur. Tuberculous ulcer presents with thin, *bluish and undermined edge*. Disease spreads more in the deeper subcutaneous plane than in the skin. Hence skin overhangs directing towards centre. It is *rounded* in shape. *Yellowish* discharge which is caseating material is common. Regional lymph nodes may get enlarged which are *matted, firm* and nontender. Study of discharge; *AFB* staining; edge biopsy; ESR; chest X-ray reveals the diagnosis. *Epithelioid cells* (modified histiocytes) are typical of tuberculosis (**Figs 2.42A to C**).

Lupus Vulgaris

Lupus means 'wolf'. It is cutaneous *tuberculosis* which occurs in young age group. Commonly it is seen in



Figs 2.42A to C: Tuberculous ulcer over chest wall and neck. Neck is the common site and is from tuberculous lymphadenitis. Note the undermined edge. Discharge study, biopsy and later antituberculous drugs are the treatment.



Fig. 2.43: Lupus vulgaris in forearm and thumb. Biopsy confirmed tuberculosis. Patient needs antituberculous drugs (Courtesy: Dr Ashfaque DNB, Surgeon, KMC, Mangalore).

face, hands and forearm (**Fig. 2.43**); starts as *typical apple-jelly nodule* with congestion of face around. It begins as superficial multiple nodules in skin which eventually forms multiple superficial ulcers with scarring, necrosis and *undermined edge*. Centre area gradually heals apparently; periphery shows active spreading disease. Often lesion extends into nose and oral cavity involving mucosa. Due to lymphatic obstruction oedema of face can occur. Long standing lupus vulgaris can turn into *squamous cell carcinoma*.

Bazin's Disease

It is also called as *Erythema induratum/Erythrocyanosis frigida*. It is localised area of fat necrosis affecting *adolescent girls*. Symmetrical purple nodules develop in the ankles and calves which eventually break down forming small, multiple, indolent ulcers with pigmented scars. It may be *due to tuberculosis*. Earlier it is thought to be due to poor blood supply of the skin around ankle, due to absence or poorly functioning ankle perforators causing low form of persistent ischaemia around ankle skin. In cold season, ankle becomes cold, bluish and tender; in warm season ankle becomes warmer, red, oedematous, painful and tender due to hyperaemia.

Traumatic Ulcer

Such ulcer occurs after trauma. It may be mechanical—dental ulcer in the margin of the tongue due to tooth injury; physical like electrical burn; chemical like by alkali injury. Such ulcer is acute, superficial, painful and tender. Secondary infection or poor blood supply of the area make it chronic and deep.

Arterial/Ischaemic Ulcer

It is common in toes, feet or legs; often can occur in upper limb digits. It is due to poor blood supply following blockage of the digital or medium sized arteries. Atherosclerosis and TAO (Thrombo Angiitis Obliterans) are common causes in lower limb. Cervical rib, Raynaud's phenomenon and vasculitis are common causes in upper limb. Ulcer initially occurs after trauma, soon becomes nonhealing, spreading with scanty granulation tissue. Ulcer is very painful, tender and often hyperaesthetic. Digits may often be gangrenous. Intermittent claudication, rest pains are common. Other features of ischaemia are obvious in the adjacent area. They are—pallor, dry skin, brittle nail, patchy ulcerations, and loss of hair. Ulcer is usually deep, destructs the deep fascia, exposing tendons, muscles and underlying bone. Dead tendons look pale/greenish with pus over it (**Figs 2.44 and 2.45**).



Fig. 2.44: Diabetic ulcer foot with ischaemia.



Fig. 2.45: Ischaemic ulcer foot. Middle three toes are already amputated because of gangrene.

Venous Ulcer (Gravitational Ulcer)

It is common around ankle (*gaiter's zone*) due to chronic venous hypertension. It is due to *varicose veins* (long saphenous vein/short saphenous vein/perforators) or *post-phlebitic* limb. Post-phlebitic limb is partially recanalised deep venous thrombosis which causes increased venous pressure around ankle through perforators. Varicose veins are common in *females*. 50% of venous ulcer is due to varicose veins; 50% is due to post-phlebitic limb (previous DVT). Pain, discomfort, pigmentation, dermatitis, lipodermatosclerosis, ulceration, periostitis, ankle joint ankylosis, talipes equinovarus deformity and Marjolin's ulcer are the problems of varicose veins and later venous ulcer. Ulcer is initially painful; but once chronicity develops it becomes painless. Ulcer is often vertically *oval*; commonly located on the *medial side*; occasionally on lateral side; often on both sides of the ankle; but *never* above the middle third of the leg. Floor is covered with pale or often without any granulation tissue when well granulated edge is sloping. Induration and tenderness is seen often in the base of an ulcer. Ulcer heals on rest and treatment; but reforms again. Scarring is common due to repeated healing and recurrent ulcer formation (**Fig. 2.46**). This *unstable scar* of long duration may lead into squamous



Fig. 2.46: Venous ulcers in both feet. Site is around ankle (gaiter's zone). There are healthy granulation tissues. It needs skin grafting and definitive procedure for varicose veins after evaluation.

cell carcinoma (*Marjolin's ulcer*) (**Fig. 2.47**). Inguinal lymph nodes (*vertical group*) are often enlarged. Ulcer often attains very large size which is nonhealing, indolent and callous.

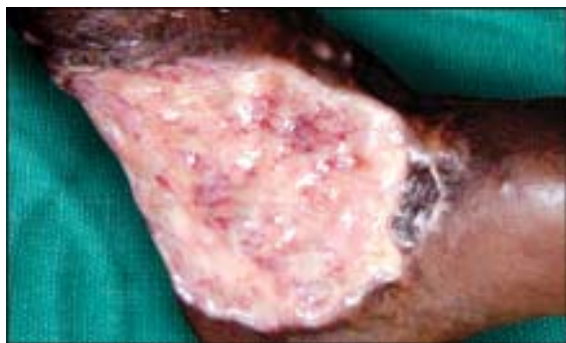


Fig. 2.47: Marjolin's ulcer can develop in a chronic longstanding venous ulcer.

Carcinomatous Ulcer (Epithelioma, Squamous Cell Carcinoma)

It arises from prickly cell layer of skin. It may initially begin as a nodule or ulcer; but later forms an ulcerative

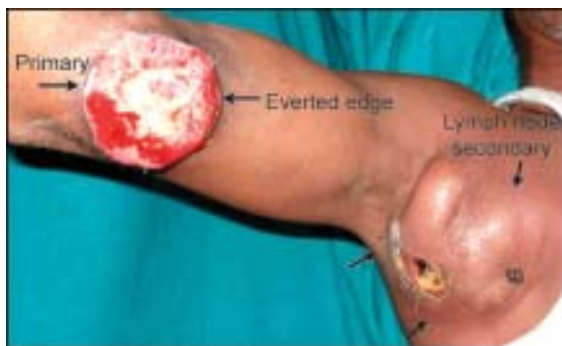


Fig. 2.48: Proliferative squamous cell carcinoma heel. Note the rolled out (everted) edge.

lesion with rolled out/everted edge (**Fig. 2.48**). Floor contains necrotic content, unhealthy (tumour) granulation tissue and blood. Ulcer bleeds on touch and is vascular and friable. Induration is felt in the base and edge. It is usually circular or irregular in shape. Initially ulcer is mobile but becomes nonmobile once it infiltrates into deeper tissues. Hard, discrete, initially mobile but later fixed regional lymph nodes are often palpable (**Fig. 2.49**). Lymph nodes can fungate eventually. Ulcer and lymph nodes are initially painless; but becomes painful and tender once there is deeper infiltration or secondary infection. Systemic spread is rare. It is a *loco-regional malignant disease*. *Verrucous carcinoma* is exophytic, locally malignant well differentiated squamous cell carcinoma without lymphatic spread. For details refer Chapter 3: Examination of Swelling.



Fig. 2.49: Squamous cell carcinoma in the arm with secondaries in the axillary lymph node. Friable tumor tissues in the floor causing bleeding after trauma. Secondaries are fixed with ulceration. It is advanced disease.

Marjolin's Ulcer (1828)

It is slow growing *locally* malignant lesion—a very well differentiated squamous cell carcinoma occurring in *unstable scar of long duration*. It is commonly seen in chronic venous ulcer scar. Often it is observed in burns scar and scar of previous snake bite. Lesion is ulcerative/proliferative. Edge may be everted or may not be. It is painless as scar does not contain nerve fibrils. It *does not* spread into lymphatics as scar is devoid of lymphatics. Induration is felt at edge and base. There is marked fibrosis also. Once lesion spreads into adjacent normal skin, it can spread into regional lymph nodes (**Fig. 2.50**).



Figs 2.50A and B: Marjolin's ulcer in the leg. It occurs in an unstable scar of long duration. It does not spread through lymphatics.

Rodent Ulcer

It is ulcerative form of basal cell carcinoma which is common in face. Ulcer shows central area of dry scab with peripheral active raised and *beaded (pearly white)* edge. Often floor is pigmented. It erodes into deeper plane like soft tissues, cartilages and bones hence the name—*rodent ulcer*. As lymphatics are blocked early in the disease by large tumour cells, it does not spread to regional lymph nodes. Blood spread is absent. It is only *locally malignant*. It is common in face; rarely it can occur over tibia,

external genitalia, mucocutaneous junction. It does not occur in mucosa. For details refer Chapter 3: Examination of Swelling (**Fig. 2.51**).



Fig. 2.51: Nodular BCC in the nose which eventually may ulcerate to form rodent ulcer.

Melanotic Ulcer

It is ulcerative form of melanoma. It can occur in skin as *de novo* or in a pre-existing mole. Ulcer is pigmented often with a halo around. Ulcer is rapidly growing, often with satellite nodules and 'in-transit' lesions. It is very aggressive skin tumour arising from melanocytes. It spreads rapidly to regional lymph nodes which are pigmented. Blood spread is common to liver, lungs, brain, and bones. It can occur in mucosa, genitalia, and eye. It is a systemic malignant disease. For details refer Chapter 3: Examination of Swelling.

Syphilitic Ulcer

Nowadays it is a rare entity. It is caused by *Treponema pallidum* bacterium. It is a sexually transmitted disease. It is named as '*Syphilis*' after a Shepherd named Syphilus who acquired the disease as was written in a poem by Francastorius of Verona. Many clinical lesions are observed in different stages of syphilis. John Hunter inoculated syphilis organism to himself to study the clinical features and effects. After 24 years of inoculation, he died at the age of 65 from rupture of syphilitic aortic aneurysm. Genital chancre (*Hard chancre, Hunterian chancre*) is painless, hard,

button like, indurated, nonbleeding ulcer; usually seen in corona or frenum of penis, often on lips, breasts and anal region; appears 4 weeks after initial infection in **first stage** of the disease (**primary syphilis**). Shotty, painless, firm, discrete groin lymph nodes may get enlarged along with genital chancre. Suppuration in these nodes will not occur. Extragenital chancres in lips and breasts show enlarged neck/axillary nodes which are inflamed, painful and often may be matted also. During **second stage** (**secondary syphilis**) white, thickened *mucous patches* appears commonly in the mouth like small, circular, superficial *snail track* ulcers. Also there appears raised, flat, hypertrophied, and warty like epithelium at mucocutaneous junctions (mouth, genitalia) called as *condyloma lata*. Generalised, shotty, hard, discrete, painless lymph nodes, epitrochlear and suboccipital lymph nodes in particular, are enlarged. *Epitrochlear nodes* are felt 1-2 cm above the medial epicondyle (It is also enlarged in Non-Hodgkin's lymphoma/NHL). Iritis, arthritis, hepatitis (massive liver in syphilis is called as *hepar lobatum*), meningitis, syphilitic osteitis with 'ivory' sequestrum, coppery red skin rash, moth-eaten alopecia are other features of second stage. In **tertiary/late stage syphilis** *gummatous ulcer* develops. It is deep, punched out, painless, nontender ulcer with wash leather slough in the floor, with 'silvery tissue paper' like scar around and occurs over the subcutaneous bones like tibia, sternum, skull, palate or other area. It also can occur in the tongue, anterior aspect of the scrotum. It is due to delayed hypersensitivity reaction with endarteritis obliterans and vasculitis. Perforation of nasal septum/palate can occur. *Clutton's joint* and *Sabre tibia* are often seen. Lymph nodes are not affected in tertiary syphilis. Neurosyphilis (*tabes dorsalis*), *aneurysm of arch of aorta* are other features of tertiary syphilis. *Tabes dorsalis* presenting as generalised paralysis of insane is often called as **late tertiary or quaternary syphilis**. Long quiescent asymptomatic period from secondary to tertiary is called as **latent syphilis**. Secondary syphilis stage shows plenty of circulating *Treponema spirochaetes* in blood whereas in tertiary stage *spirochaetes* are less or absent in circulation.

Soft Chancre/Soft Sore/Ducrey's Ulcer/ Chancroid/Bubo

These multiple irregular genital ulcers that appear 3 days after infection with *Haemophilus ducreyi* as a *venereal* disease. They are acute painful, tender, non indurated ulcers. Floor shows yellowish slough with purulent discharge. Edge is oedematous and inflamed. Acute regional lymphadenitis with suppuration presenting as tender, soft or firm swelling is common. Such soft fluctuant inguinal swelling is termed as **bubo**. It differs from **climatic bubo/tropical bubo** which is due to *lymphogranuloma inguinale*, a *venereal* spreading organism (LGV, Chlamydia type L1, 2, 3). In LGV, *primary genital stage* lesion is small and painless and commonly unnoticed. *Secondary stage* lesion develops in 2 weeks. In males inguinal lymph nodes; in females intrapelvic and pararectal nodes are involved. Suppuration of inguinal nodes occurs eventually leading into discharging sinuses. *Frei intradermal test* becomes positive in 6 weeks and remains positive for life time. In *tertiary stage*, eye, joint, meninges may involve after many years. Repeated chronic inflammation, lymphatic blockage, scarring can cause rectal stricture and vulval elephantiasis (*esthiomene*) in females.

Other Ulcers

Ulcers can occur in various parts like over shin, legs, feet, face, chest wall in various diseases like anaemia, polycythaemia, sickle cell disease, hereditary spherocytosis, lukaemia, vasculitis, autoimmune diseases like rheumatoid arthritis, Paget's disease of bone (deep, nonmobile, fixed to bone; common in tibia), ulcerative colitis, etc. *Treponema pertenue* causing Yaws can have multiple painless ulcers in leg and feet due to bare foot walking (organism enters through abrasion) which heals spontaneously with a tissue paper like scar. Poor hygiene and dressings can cause multiple, small, red often scabbed *Staphylococcus aureus* ulcers in the skin of the leg and feet which is often recurrent and disturbing. Traumatic staphylococcal ulcer is often seen in the shin which may become chronic and deep, and is seen in footballers—'Footballer's ulcer'.

3

Examination of a Swelling/Lump

Swelling/lump denotes enlargement or protuberance in any part of the body, due to congenital/inflammatory/traumatic or neoplastic causes. Often in areas like abdomen, word ‘mass’ is used to denote a swelling. The word ‘mass’ is usually used in a large swelling where its extent is difficult to estimate. In breast, word ‘lump’ is commonly used. There is no clear cut difference in each of these terminologies (as by meaning all are same), but *purely on clinical grounds* it is used in different places like for example ‘swelling’ in the skin (swelling means an eminence or elevation); ‘lump’ in the breast (lump means something hard or solid); ‘mass’ in the abdomen. Any of these can be often visible and palpable or may be only palpable but not visible.

History

History of Present Illness

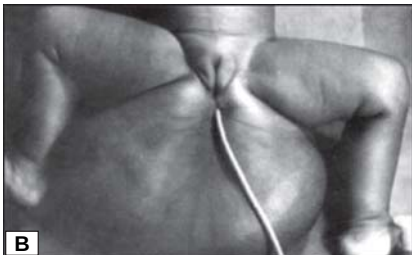
Duration

It is important to note the duration of all swellings.

Swelling which has been present since birth could be *congenital* like meningocele. Swelling of short duration associated with pain may be of *inflammatory* origin. Acute inflammatory swelling will be of short duration with severe pain. Chronic inflammatory swellings often have long duration with mild pain. *Benign tumours* are usually painless swelling of long duration. *Malignant tumours* present as swellings of short duration, rapidly enlarging, initially painless (but can be painful later). Patient may not be aware of the existence of a painless swelling for a long time. Often patient will not give much importance to a painless swelling (**Figs 3.1A to C and 3.2**).

Mode of Onset and Progress

It is very important to take the history regarding the mode of onset of the swelling. Swelling whether occurred after trauma (example—haematoma) or spontaneously. It is important to note the rate of progress,



Figs 3.1A to C: Sacrococcygeal teratoma in newborn infants and also X-ray of the same condition.



Fig. 3.2: Spina bifida—a congenital anomaly of spine presenting as swelling. Failure of fusion of posterior part of the spine is called as *spinal dysraphism*. It can be spina bifida occulta or spina bifida aperta. Meningocele, meningo-myelocele, syringomyelocele, myelocele are different types of spina bifida aperta.

whether rapid or slow, malignant swellings progresses rapidly whereas benign swellings progress slowly. Sudden haemorrhage in a swelling can cause increase in its size rapidly in minutes to hours. Sarcomas may progress rapidly in weeks. Swelling that shows recent rapid progress in size means probably benign lesion is turning into malignancy. Swelling which eventually shows reduction in size is probably of inflammatory origin. Certain swellings may be stationary—status quo, i.e. neither progressive nor regressive (**Figs 3.3 and 3.4**).

Site of beginning of the swelling and its eventual progression is also often an important history to find out the anatomical origin of the swelling. Side and exact site should be asked. Size and shape of the swelling at the time of initial observation should be asked.

Number of swellings patient has observed and which swelling appeared first and next in order should be asked. Progression of each should be clarified.

Pain

When pain started? Detail history of location of pain/type of pain/severity/whether it interferes with work or not is to be noted. Inflammatory conditions are painful whereas malignant conditions are painless to begin with but later becomes painful. Infiltration into the nerves, soft tissues; ulceration; necrosis or inflammation may be the cause of pain in malignancy



Fig. 3.3: Abscess on chest wall in a patient who has undergone mastectomy earlier for carcinoma of breast. Patient was on chemotherapy and presented with abscess in the region of acute onset and short duration with pain, fever, tenderness, redness and swelling.



Fig. 3.4: Haematoma ear. It is subperichondrial haematoma, which usually occurs in boxers, wrestlers and rugby players can also occasionally occur spontaneously. Presents as discoloured, doughy soft swelling with feeling of heaviness and discomfort. Fluctuation may be absent as there may be complete clotting of extravasated blood. It resolves very slowly. Often there is oedema of adjacent part of the ear. Pain is usually absent. Repeated multiple subperichondrial haematomas of ear leads to cauliflower ear which is unsightly, deforming and often may lead into cartilage necrosis and destruction. Bleeding disorders should be thought of if haematoma is of spontaneous onset.

eventually. Rapid enlargement of malignant tumour or haemorrhage also can cause pain in malignancy. Pain is usually over the swelling but often it can be deep seated pain or *referred pain* towards different place away from the swelling. In a large swelling pain may be only over certain part of the swelling.

Nature of the pain is important to be noted. Pain may be *throbbing* in acute inflammation or suppuration; *burning* in inflammatory conditions or neurological causes like Herpes Zoster infection; *aching*; *stretching*; *distending*; *deep seated*; *sharp*; *vague*; *stabbing*, etc.

Presence of Fever

Fever may be present in inflammatory conditions. Pyogenic abscess, acute lymphadenitis are associated with fever, often of high grade. Certain malignancies also can present with fever at later stage like in Hodgkin's lymphoma or renal cell carcinoma.

Presence of Other Lumps

Multiple neurofibromatosis, lipomatosis, multiple abscesses in the body, generalised lymphadenopathy of any cause (Lymphomas) are the examples of multiple swellings in the body.

Secondary changes in the swelling like ulceration/fungation/bleeding has to be noted.

Loss of function of part or as a whole. Patient with cold abscess may show spinal pathology with alteration in limb movements, sensation, etc. Swellings adjacent or from the joint will show impaired joint function.

Loss of weight and decreased appetite may signify that swelling is related to malignant condition and also probably advanced.

Past History

History of previous surgery for similar swelling at the same site or different site has to be asked for. Neurofibroma even though once excised often may occur at some other place in the body. Incompletely removed earlier benign lesion, either cyst or tumour or if the lesion is a malignant one then recurrence can occur at the same site.

Personal History

Personal history of alcohol consumption/smoking/tobacco chewing/history of sexual contact/dietary

habits are also important. Altered appetite or weight loss can also be mentioned under personal history.

Family History

Family history suggestive of similar swellings is important. Neurofibromatosis is often familial. History of tuberculosis among the family members may be relevant in cold abscess. Certain malignancies can run in families.

General Examination

Detailed general examination is very essential. Anaemia/oedema/jaundice/clubbing/lymphadenopathy/radial pulse/blood pressure/raise in temperature/attitude of the patient/nutritional assessment by skin texture, subcutaneous fat, weight, body mass index/any other relevant findings should be mentioned. Cachexia signifies advanced malignancy or tuberculosis. Bone tumours, malignant tumour infiltrating nerves can alter the attitude of the limb. Increased pulse rate and fever suggests swelling with inflammatory pathology.

Local Examination

Inspection

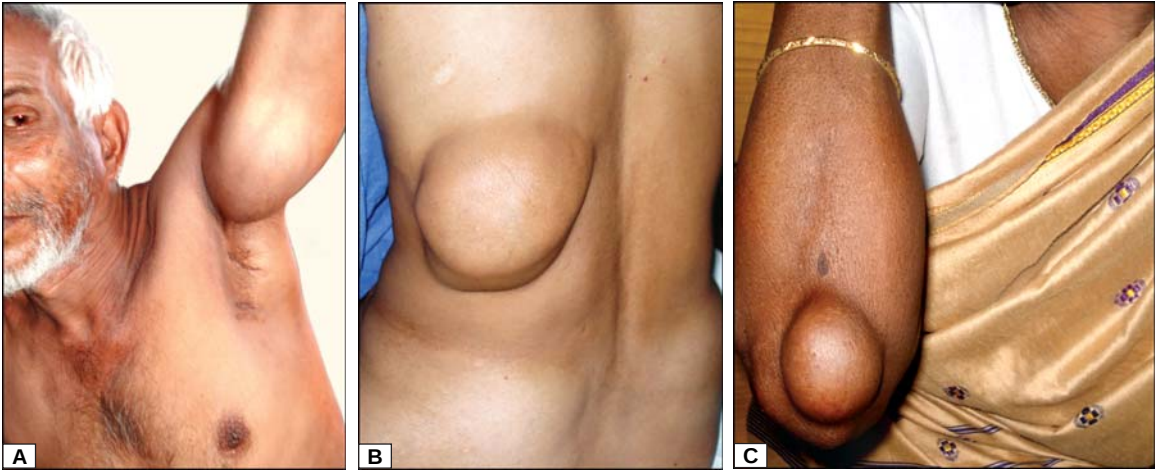
Location, Size and Shape of the Swelling

Exact anatomical location of the swelling and its size is noted. Its shape—globular or haemispherical or oval or pear-shaped or irregular or kidney shaped/diffuse or well localised is noted. As deeper part of the swelling is not seen, it is not possible to say a swelling as 'circular' but can be told as spherical (**Figs 3.5A to C**). Vertical and horizontal dimension should be assessed and should be measured using a measuring tape. Site of the swelling is mentioned from a fixed bony prominence like tibial tubercle, sternal angle, angle of the mandible, etc. (**Fig. 3.6**).

Dermoid cysts occur in midline/outer canthus of eye/or any embryonic line of fusion. Lipoma can occur anywhere in the body.

Colour of the Swelling

Blue colour of haemangioma/black colour of naevus or melanoma/blue colour of ranula are often diagnostic. Redness over the swelling suggests inflammation (**Fig. 3.7**).



Figs 3.5A to C: Swelling should be inspected properly for its exact anatomical location, shape, size and extent.



Fig. 3.6: In a parotid swelling, raise of ear lobule is an important finding which should be observed during inspection.

Surface over Swelling

The surface may be smooth/irregular (papilloma)/nodular/cauliflower like (squamous cell carcinoma)/lobular.

Number of the Swellings

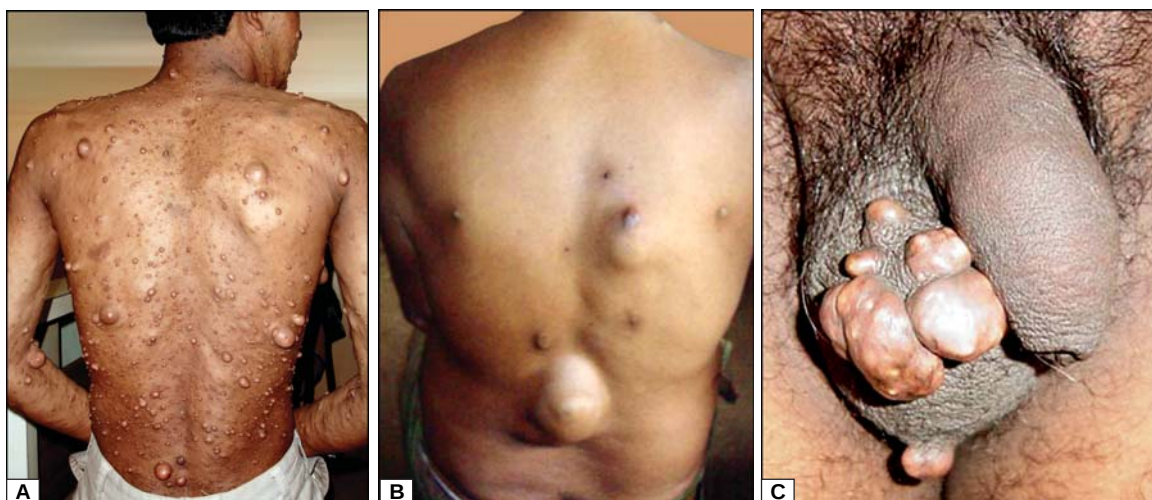
Neurofibromas and sebaceous cysts can be multiple. Dermoid cyst is usually single (**Figs 3.8 and 3.9A to C**).



Fig. 3.7: Sebaceous cyst face which is infected. Redness is well seen.



Fig. 3.8: Postauricular dermoid. Dermoid is usually a single swelling.



Figs 3.9A to C: Neurofibromas and sebaceous cysts can be multiple. In scrotum multiple sebaceous cysts are common.

Edge of the swelling whether well-defined or ill-defined/whether pedunculated or sessile should be looked for.

Pulsation over the Swelling

Arterial swelling has got *expansile pulsation* (It is checked by keeping two fingers over the swelling during palpation). Swelling which is very close to artery or adherent to it also can show pulsation but it is *transmitted pulsation*. On inspection it is possible only to tell whether swelling is pulsatile or not.

Presence of expansile impulse on coughing signifies hernia or communication into the deeper cavity like abdomen or thorax or cranium (**Fig. 3.10**).

Skin over the Swelling

Skin over the swelling should be inspected.

Skin over the swelling may be tense, glossy with prominent veins as in sarcoma and malignancy. It is red oedematous in inflammatory swellings. Pigmentation, ulceration/fungation/discharge from ulcer/bleeding from the fungation should be inspected. Bluish colour is seen over the skin in haemangioma. Black punctum over the summit of the swelling suggests sebaceous cyst. In sarcoma skin will be tense with dilated veins over the surface. *Peau d 'orange* over the swelling is due to cutaneous lymphoedema following blockage of cutaneous lymphatics usually



Fig. 3.10: In a swelling related to cavities like thorax, abdomen or cranium, expansile impulse on coughing should be checked to identify the possibility of intracavitary extension.

by malignant cells. It is commonly seen in carcinoma breast.

Scar if present: Its size, features whether healed by primary intention or secondary intention should be

mentioned. Scar may be linear and regular/broad, puckered and irregular has to be noted (**Figs 3.11 to 3.13**).



Fig. 3.11: Recurrent soft tissue tumour (sarcoma) thigh. Note the scar of previous surgery. This scar has healed by primary intention. It is a linear, smooth and supple scar.



Fig. 3.12: Sarcoma right chest wall. Note the swelling with dilated veins on the surface.

Movements of the Swelling/Mass

Upper abdominal masses like from liver, gallbladder, stomach, spleen *move with respiration*.

Thyroid swellings, thyroglossal cyst, subhyoid bursa, pre-tracheal lymph nodes which are attached to trachea/larynx *move with deglutition*.

Thyroglossal cyst also *moves with protrusion of tongue* due to its relation through the thyroglossal tract



Fig. 3.13: Sebaceous cyst surface-punctum and content sebum is extruding through the punctum.

which is attached to the base of the tongue—*foramen caecum*.

Falling forward of the lump like in breast should be looked for.

Inspect the local area as well as distally especially when swelling is in the limbs for pressure effects and wasting. Wasting should be confirmed by proper measurement of the part from equal distance from a bony point.

Palpation

It is done properly to define the swelling anatomically and also to find out the nature of the content and its pathology.

Local Raise of Temperature

Local raise of temperature is checked using *back of the fingers* which is more sensitive than palmar aspect (**Fig. 3.14**). The temperature should be checked in the beginning of palpation as in later part of palpation swelling may feel apparently warmer due to manipulation. It may be due to inflammation (infection) or due to tumours with increased vascularity. Sarcoma is warmer; cellulites, pyogenic abscess are warm. Cold abscess (due to tuberculosis) is not warm as there are no signs of acute inflammation. But secondary infection in a cold abscess can make it warm.

Tenderness

Tenderness is checked while palpating the swelling by observing the face of the patient. Patient expresses



Fig. 3.14: Back of the fingers is used to check the local raise of temperature.

the tenderness. Inflammatory conditions are tender. Neoplastic conditions are initially non-tender but later can become tender. Tenderness should be elicited gently.

Size

Size is measured using tape (vertical in cm X horizontal in cm) (**Fig. 3.15**); shape is confirmed and extent of the entire swelling and its anatomical location should be mentioned properly.

Edge or Margin

Edge or margin of the swelling can be well-defined (distinct) or ill-defined (indistinct). It is ill-defined in acute conditions and deep swellings. It is well-defined in superficial swellings. Margin may be irregular in malignancy and may be regular in benign swellings.



Fig. 3.15: Swelling should be measured using a measuring tape or scale. Usually the measurement is described in centimeter. First, vertical measurement is mentioned, later horizontal measurement.

Edge of the swelling is examined using pulp of the index finger. Erosion of the margin into the deeper plane like bone is also checked. Dermoid cyst commonly shows erosion into the bone. In lipoma margin slips away from the finger—*slip sign* (**Fig. 3.16**). In sebaceous cyst margin gets yielded by the finger.



Fig. 3.16: Typical *slip sign* is positive in lipoma.

Surface of the Swelling

It is done with the palmar surface of the fingers. It may be smooth like in a cyst/nodular in lymph nodes/lobular in lipoma/matted in tuberculous nodes/irregular in carcinoma. It may be variable and if so should be mentioned which part is smooth and which is nodular (**Fig. 3.17**).

Consistency

It may be *very soft* (like jelly)/*soft* (like consistency of lip/relaxed muscle)/may be *firm* (like consistency of nose/contracted muscle)/may be *hard* (like consistency of forehead). Lipoma, cystic swellings, abscess are soft. Fibromas, neurofibromas, certain nodal enlargements are firm. Chondroma, osteomas are *bony hard*. Malignant swellings are *stony hard*. Variable consistency may be observed in one swelling. In such occasion which area is soft, and which area is firm or hard should be confirmed properly. Variability may be due to tumour necrosis/inflammation. Swelling



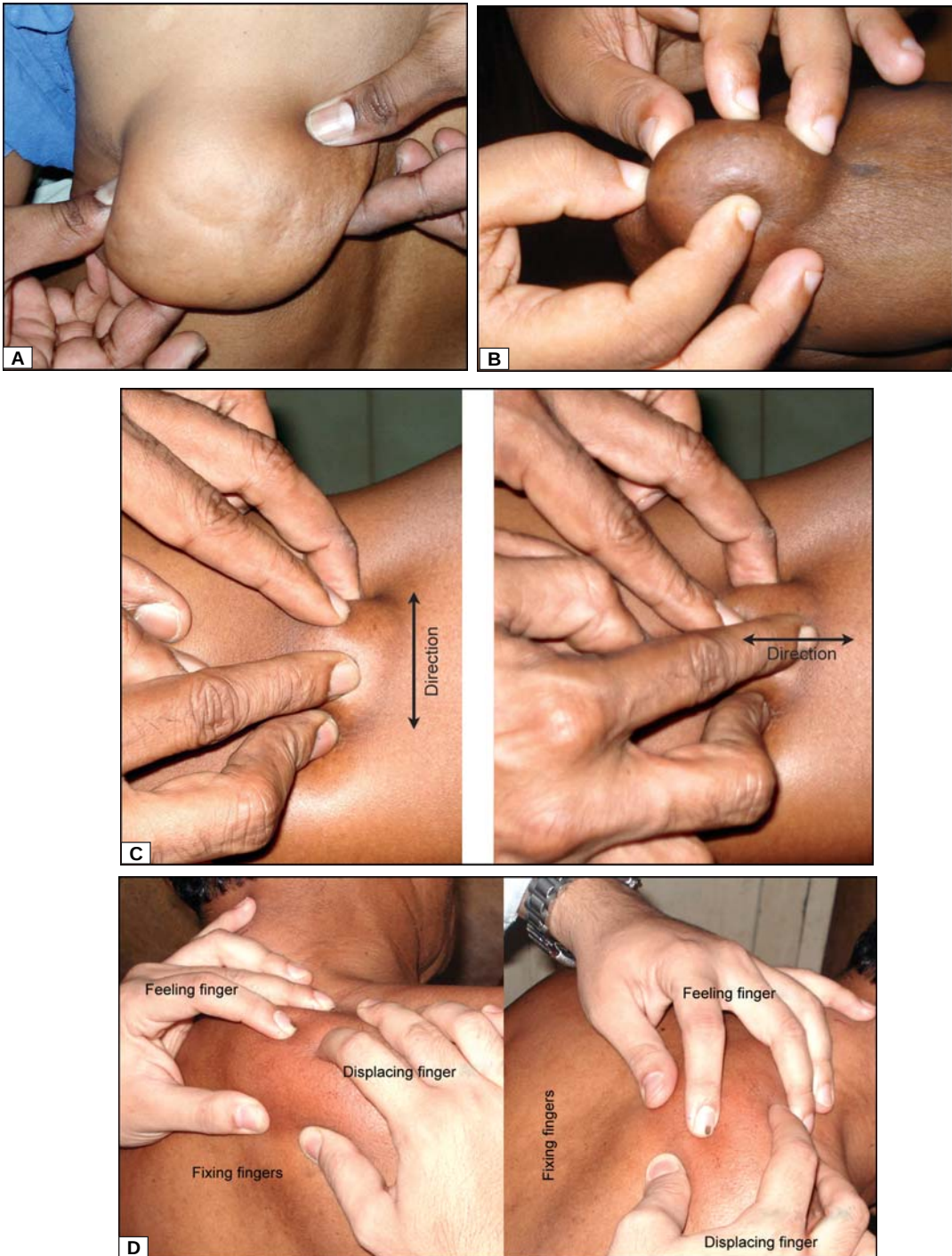
Fig. 3.17: Large lipoma over buttock which is well localised, smooth, soft and lobulated.

like sebaceous cyst or dermoid cyst which contains pultaceous (porridge like) material or putty like material gets *moulded*.

Fluctuation

Swelling is usually fixed by holding with both thumbs and middle fingers. With the index finger of one hand one side of the swelling is pressed and index finger of the other hand placed diagonally on the opposite side feels fluid movement and also a raise. Procedure is repeated in perpendicular direction to confirm fluctuation (two right angle planes). Finger used to press the swelling is called as *displacing finger* and finger that is used to feel (which is kept as passive) is called as *feeling finger*. This is **standard fluctuation**. Positive fluctuation signifies presence of fluid. Thumb and forefingers of one hand can be used to fix the swelling and fingers of the other hand can be used to displace and feel the fluid. Examples are hydrocele, cysts, etc. (Note: Often muscle gives fluctuation like feeling when elicited in one direction but not in two perpendicular directions) (**Figs 3.18A to D**).

In swelling which cannot accommodate two fingers to do standard fluctuation test, margin of the swelling is fixed using two fingers (index and ring) and using middle finger summit/centre of the swelling is pressed/indented to feel displacement of the fluid/yielding sensation. This test is called as **Paget's test** of fluctuation (**Figs 3.19A and B**).



Figs 3.18A to D: Swelling should be fixed before eliciting the fluctuation. Fluctuation cannot be elicited in intra-abdominal swelling as it cannot be fixed. It should be done in two perpendicular directions. With one finger swelling is pressed to displace the fluid content and its movement is felt with other finger placed.



Figs 3.19A and B: Paget's test is done for a small swelling to elicit fluctuation.

Fluctuation may be present in a cystic swelling which contains fluid with two components on either sides of an anatomical barrier (across an anatomical barrier). It is called as **cross fluctuation**. Ranula (across mylohyoid muscle), psoas abscess (across inguinal ligament), compound palmar ganglion (across flexor retinaculum), bilocular hydrocele (across a band or superficial inguinal ring) are cross-fluctuant.

Sense of fluctuation may be elicited in lipoma, myxoma and vascular swellings.

Transillumination Test

When light is illuminated over the swelling it transmits light through it. It is called as transillumination/

translucency. It is positive means swelling illuminates to light and also means it contains clear fluid. It is negative when it contains blood, pus, pultaceous material. Torch light is placed on one side of the swelling and illumination is observed on the diagonally opposite side using a rolled paper or rolled X-ray. Lymph cyst, cystic hygroma, ranula, meningocele, hydrocele are transilluminant swellings (**Figs 3.20 to 3.22B**).

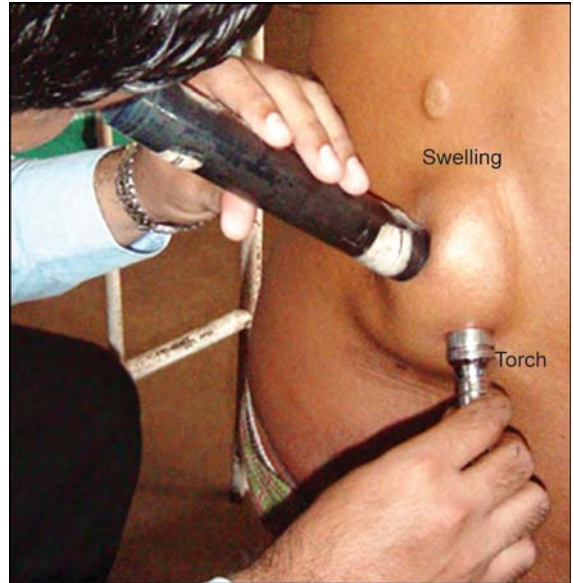


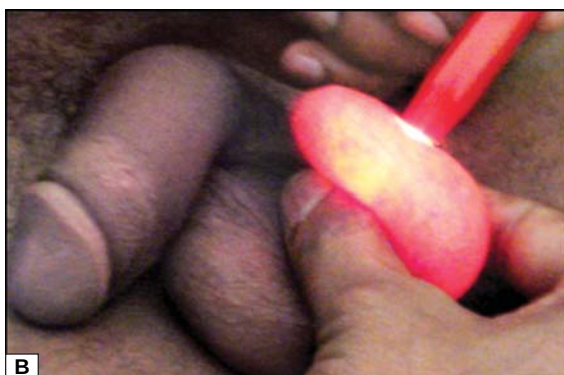
Fig. 3.20: Transillumination should be elicited using pen torch and dark visualisation tube (folded X-ray tube/scotoscope). All fluctuant swelling should be checked for transillumination.



Fig. 3.21: Brilliantly transilluminating swelling. While checking transillumination, transillumination of normal skin should be carefully eliminated.

Swellings which are brilliantly transilluminant

- Ranula
- Cystic hygroma and lymph cyst
- Hydrocele
- Epididymal cyst (*Chinese-lantern pattern*)
- Meningocele
- Hydrocele of the canal of Nuck



Figs 3.22A and B: In hydrocele transillumination should be checked by placing torch and tube in opposite directions (side-to-side) in the front aspect of the swelling. It should not be carried out from front to back as testis will prevent light to pass and make transillumination test improper.

Reducibility

When swelling is pressed gets reduced completely and disappears is said to be reducible swelling. Hernia is reducible.

Compressibility

Swelling on pressure reduces in size *only partially but will not disappear completely* and on releasing the pressure swelling again *comes back to its original size and shape immediately*. Usually vascular and lymphatic swellings are compressible, e.g. haemangioma, lymphangioma.

Pulsatility

Two fingers are placed over the swelling with adequate gap between two fingers. If fingers over the swelling are raised and separated with each beat of the artery it means pulsation is *expansile*. If fingers are only raised but not separated from each other then pulsation of the swelling is said to be *transmitted*. Pure arterial swelling like aneurysm shows expansile pulsation (**Fig. 3.23**). Swelling which is close to the artery may show pulsation because of its close proximity and it is only transmitted pulsation. Pseudocyst in the abdomen shows transmitted pulsation because of its close proximity to aorta.



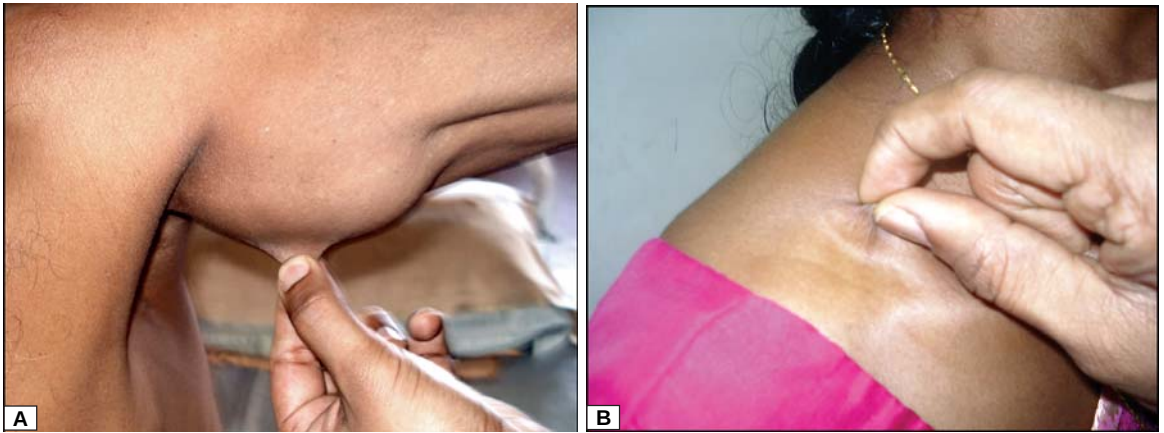
Fig. 3.23: Aneurysm in the elbow which can mimic abscess. Aneurysm shows expansile pulsation.

Fixity to the Skin

Mobility of the skin over the swelling is checked or skin over the swelling is pinched to confirm whether skin is free or attached to swelling underneath (**Figs 3.24A and B**). Sebaceous cyst has adherent skin over the summit with a punctum (70%) often present. In dermoid cyst skin is always free. In lipoma skin is usually free. In neurofibromas skin may be adherent, but depends on from which nerves neurofibroma arises, whether from deeper plane or from cutaneous nerves.

Fixity to Deeper Structures

If swelling is freely mobile it could be in subcutaneous plane (**Figs 3.25A to E**). Lipoma, sebaceous cyst, often neurofibroma are subcutaneous swelling.



Figs 3.24A and B: Skin over the swelling should be pinched/held to check whether swelling is adherent to skin or not.

If swelling is adherent to muscle underneath, then when muscle is contracted against resistance mobility of the swelling is restricted but it becomes more prominent. When muscles relaxes swelling will be mobile.

If swelling is arising from the muscle or deep to muscle then size of the swelling decreases (less prominent) when muscle is contracted. Again mobility which is present initially will disappear completely during contraction of the muscle. Disappearance occurs much more significantly in swelling which is deeper to the muscle (**Fig. 3.26**).

Swellings arising from vessels or nerves will move only in horizontal direction/perpendicular to the line of nerve but will not show any mobility in longitudinal direction, e.g. neurofibroma, aneurysm.

Swelling arising from the bone is hard and absolutely fixed and cannot be moved separately from the bone (**Fig. 3.27**).

Methods of Contractions of Different Muscles Against Resistance

Figures 3.28 to 3.44 describe contraction methods of various types of muscles against resistance.

Examination distally and proximally is essential to see *pressure effects*; wasting. Pressure effects may be on adjacent artery (absence/feeble pulse; ischaemic changes); nerves (wasting; paresis; altered sensation; deformities, etc); over the bone (pressure erosion of the adjacent bone like in aneurysm, dermoid cyst, malignancy, etc).

Percussion over the swelling in relevant areas like hernia should be done. Laryngocele in the neck is resonant. Abdominal mass should always be percussed. Often tenderness may be elicited by percussion (**Fig. 3.45**).

Auscultation

It is done to look for bruit over the swelling like in A-V malformation, arterial stenosis, aneurysms. *Machinery murmur* is heard in AV fistulas.

Joints above and below the swelling should be examined both for active and passive movements.

Regional Lymph Nodes

Regional lymph nodes should be examined for significant enlargement. Other groups/proximal groups should be examined in relevant/systemic clinical indications (**Figs 3.46A and B**).

Relevant Systemic Examination

Systemic examination is a must like respiratory (to look for pleural effusion, consolidation, cavity), cardiac, skeletal (bones and joints for osteomyelitis, gibbus, kyphosis, scoliosis, deformities) and abdomen (for mass, fluid).

Proper diagnosis of the swelling should be given.

Relevant Investigations

FNAC, U/S of part, CT scan, MRI for bony and joint swellings, angiography and Doppler in vascular swellings, biopsy in soft tissue sarcomas.



Figs 3.25A to E: Mobility of swelling should be checked to find out the plane of the swelling.

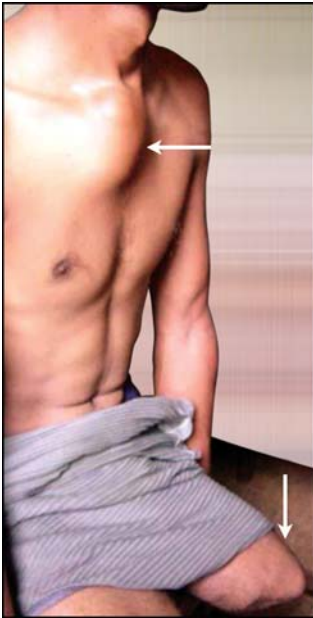


Fig. 3.26: Bony swelling in sternum which is nonmobile. In this patient it is secondaries from osteosarcoma of lower femur (thigh amputated).



Fig. 3.27: Secondaries in the skull in a patient with primary in the thyroid (follicular carcinoma). Follicular carcinoma of thyroid causes localised, warm, vascular, pulsatile, smooth, hard/soft, (nonmobile) secondaries in skull.



Figs 3.28A and B: Contraction of extensors of ankle.



Fig. 3.29: Contraction of flexors of the ankle.



Fig. 3.30: Contraction of hamstring muscles.



Fig. 3.31: Contraction of gluteus medius muscle.



Fig. 3.32: Contraction of gluteus maximus muscle.



Fig. 3.33: Contraction of adductors of thigh.



Figs 3.34A and B: Contraction of quadriceps femoris.



Fig. 3.35: Contraction of wrist flexors.



Fig. 3.36: Contraction of wrist extensors.



Fig. 3.37: Contraction of triceps brachii muscle.



Fig. 3.39: Contraction of latissimus dorsi muscle.



Figs 3.38A and B: Contraction of biceps brachii muscle.



Figs 3.40A and B: Contraction of trapezius muscle.



Fig. 3.41: Contraction of serratus anterior muscle.



Fig. 3.42: Contraction of pectoralis major muscle.



Figs 3.43A to C: Contraction of sternomastoid muscles both sides together and each side independently.



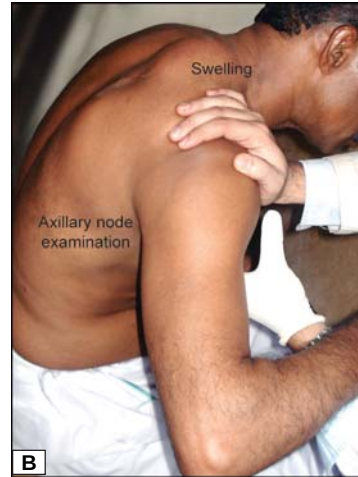
Fig. 3.44: Contraction of abdominal wall muscles.



Fig. 3.45: Swelling should be auscultated for any bruit.

Differential Diagnosis

Swellings may be **congenital/traumatic/inflammatory/neoplastic**. It may be benign or malignant. In malignancy it may be early or advanced. First **anatomical diagnosis** of the swelling should be made by clinical methods and proper analysis. It means from which anatomical structure the swelling is arising from (Fig. 3.47). Anatomical diagnosis can be by various clinical methods like movements, relation to muscle, plane of the swelling. Then **pathological diagnosis** is made out by examining the surface, consistency, fluctuation, transillumination, tenderness, warmth. When features elicited are not suitable for one diagnosis, it is not possible to give a single diagnosis. Then differential diagnoses should be given. While giving differential diagnosis, clinical features which



Figs 3.46A and B: Relevant regional lymph node examination should be done to look for palpable significant nodes.



Fig. 3.47: Epignathus. It is a type of growth anomaly seen in neonates wherein growth from the base of skull protrudes into the mouth.

correlate to most possible condition should be given as first possible diagnosis; like that second; third, etc. *It is not necessary to give every condition as differential diagnosis.* Only conditions relevant to those clinical features should be given as differential diagnosis. Congenital conditions are—haemangiomas; dermoid cyst, etc. Cellulitis, abscess, boil, carbuncle are inflammatory conditions. Neoplasms can be benign or malignant. Lipoma, papilloma, neurofibromas are examples of benign swellings. Malignant skin tumours, sarcomas are malignant tumours. Other swellings like sebaceous cyst, keloid, pyogenic granuloma are also important.

Different conditions which are discussed here are:

Cysts	Cellulites
Sebaceous cyst	Erysipelas
Dermoid cyst	Abscess
Lipoma	Furuncle
Neurofibroma	Carbuncle
Schwannoma	Condyloma
Papilloma	Moles
Seborrheic keratosis	Basal cell carcinoma
Solar keratosis	Squamous cell carcinoma
Pyogenic granuloma	Malignant melanoma
Fibroma	Sarcoma
Haemangiomas	
Lymphangiomas	
Keloid	
Keratoacanthoma	
Corn	
Callosity	

Cysts

Cyst is a collection of fluid in a sac lined by epithelium or endothelium. Word meaning of cyst is '**bladder**' (Greek). In **true cyst**, cyst wall is lined by epithelium or endothelium. If infection occurs cyst wall will also be lined by granulation tissue. Fluid is usually serous or mucoid derived from the secretion of the lining. In **false cyst**, cyst does not have epithelial lining. Fluid collection occurs as a result of exudation or degeneration. *Examples:* Pseudocyst of pancreas, wall of cystic swelling in tuberculous peritonitis, cystic degeneration of tumour, after haemorrhage in a haematoma red cells are lysed, get absorbed and fluid remains as a false cyst. '**Apoplectic cyst**' is formed in brain as a result of ischaemia causing collection of fluid.

Classification of Cysts

- a. Congenital cyst:** *Dermoids:* Sequestration dermoid; *Tubulodermoids:* Thyroglossal cyst, postanal

dermoid, ependymal cyst, urachal cyst; *Cysts of embryonic remnants:* Cysts from paramesonephric duct and mesonephric duct; Cysts of urachus and vitelointestinal duct.

- b. Acquired cysts:** *Retention cysts:* They are accumulation of secretion of a gland due to obstruction of a duct. Examples: Sebaceous cyst, Bartholin cyst, cyst of pancreas, cyst of parotid, breast, epididymis. *Distention cyst:* Lymph cyst, ovarian cyst, colloid goitre. *Exudation cyst:* Bursa, hydrocele.
- c. Cystic tumours:** Dermoid cyst of ovary, cystadenomas.
- d. Traumatic cyst:** Due to trauma, haematoma occurs usually in thigh, loin, and shin. It eventually gets lined by endothelium containing brown coloured fluid with cholesterol crystals.
- e. Degenerative cyst:** Due to cystic degeneration of a solid tumour (due to necrosis of tumour).
- f. Parasitic cyst:** Hydatid cyst, trichiniasis, cysticercosis.

Clinical features of a cyst: Hemispherical swelling which is smooth, fluctuant, nontender, well-localised. Some cysts are transilluminant. Presentation varies depending on its anatomical location.

Effects of a cyst: *Compression* to adjacent structures: choledochal cyst compressing over the CBD; *infection;* sinus formation; *haemorrhage;* *torsion* like in ovarian cyst; *calcification;* cachexia: in malignant ovarian cyst patient goes for severe cachexia.

Dermoids

Types

- a. Sequestration dermoids:** It occurs at the line of embryonic fusion due to inclusion of epithelium beneath the surface which later gets sequestered forming a cystic swelling in the deeper plane.

Common sites are: Forehead; external angular dermoid; root of nose; post-auricular dermoid; sublingual dermoid; in the ear; anywhere in midline or in the line of fusion. Dermoids occurring in the skull may extend into the cranial cavity. When it occurs as external angular dermoid, it extends into the orbital cavity. Or it can extend into any cavity in relation to its anatomical location (e.g. thorax, abdomen). Dermoid cyst contains putty like desquamated material. It is lined by both dermal and epidermal components.

External angular dermoid: It is a sequestration dermoid situated over the external angular process of the frontal bone (frontozygomatic suture). Outer extremity of the eyebrow extends over some part of the swelling. This typical feature differentiates it from the swelling arising from the lacrimal gland. It may extend into the orbital cavity also (**Fig. 3.48**).



Fig. 3.48: External angular dermoid.

Internal angular dermoid: It is a sequestration dermoid cyst in central position at the root of the nose. Dermoid cyst in scalp may lie purely in the scalp or may cause a defect in the skull with attachment to dura or may be partly intracranial and partly extracranial with a stalk between the two parts or very rarely purely intracranial lying deep to skull and outer to dura but attached to it (**Fig. 3.49**).

Clinical features: Painless swelling in the line of fusion, presents in the second or third decade onwards, which



Fig. 3.49: Internal angular dermoid.



Fig. 3.50: Dermoid in the ear. It arises due to sequestration at the fusion line of one of the six developmental ear tubercles. (Each ear develops from six ear tubercles).



Fig. 3.51: Midline dermoid.

is smooth, soft, nontender, fluctuant (*Paget's test positive*, i.e. swelling is fixed with two fingers and summit is indented to get yielding sensation due to fluid), nontransilluminating, with free skin often adherent to the deeper plane (**Fig. 3.50**). There will be resorption **and indentation** of the bone beneath. Impulse on coughing may be evident if there is intracranial extension. It should be differentiated from lipoma and sebaceous cyst. Slip sign and free mobility are features of lipoma. Skin is adherent in sebaceous cyst often with a punctum (**Fig. 3.51**). X-ray part or CT scan is often needed to evaluate its deeper extent.

Submental dermoid: It is a congenital sequestration dermoid occurs during fusion of 1st and 2nd branchial

arches. It is deep to deep fascia of neck. It presents as soft, cystic, fluctuant, nontransilluminating, swelling in midline in submental region which does not move with deglutition nor moves while protruding the tongue out. It should be differentiated from thyroglossal cyst, cold abscess from submental lymph nodes or sebaceous cyst.

b. Tubulodermoids: It arises from the embryonic tubular structures. Examples includes—Thyroglossal cyst; ependymal cyst; postanal dermoid.

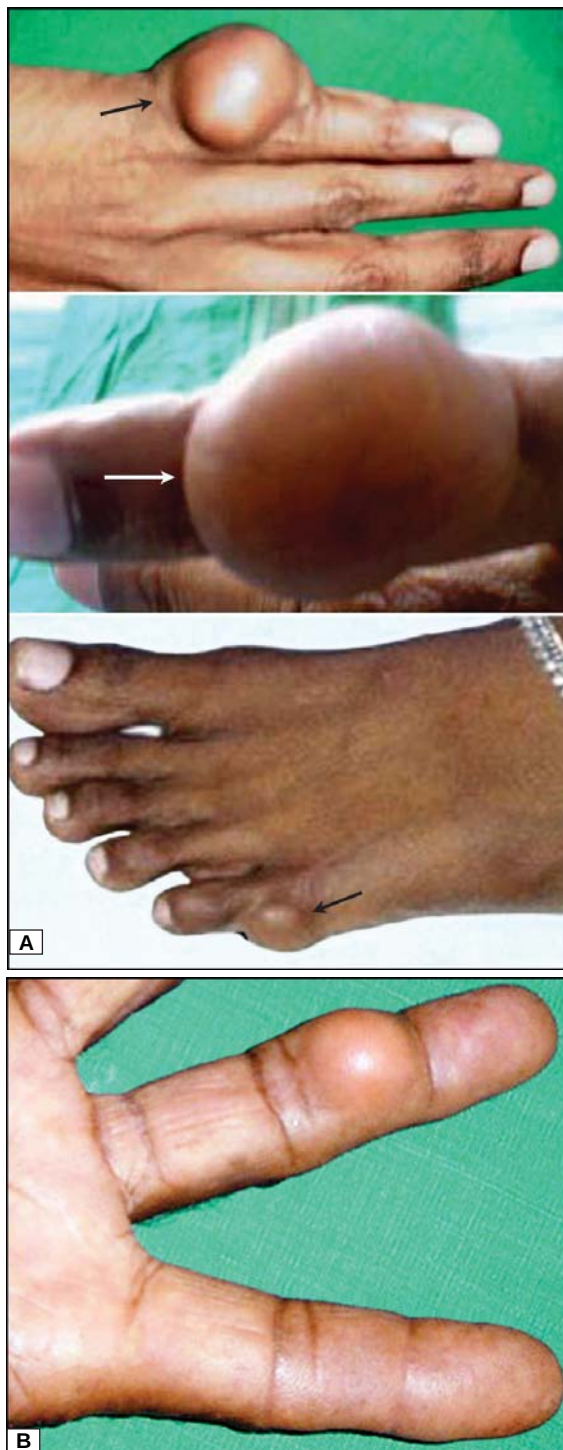
c. Implantation dermoid: Due to minor pricks or trauma, epidermis gets buried into the deeper subcutaneous tissue which causes reaction and acquired cyst formation (trauma is often forgotten). It is common in fingers (common in tailors), toes and feet (**Figs 3.52A and B**). It is slowly progressive swelling after a trauma which is smooth, soft, mobile, tensely cystic, nontransilluminating and is adherent to skin. It contains only squamous epithelium without hair follicle/sweat glands/sebaceous glands. It can cause infection, rupture or pressure effects on digital nerves.

d. Teratomatous dermoid: It arises from all germinal layers ecto, meso and endoderms. It occurs in ovary (**Fig. 3.53**), testis, retroperitoneum, mediastinum. It contains hairs, teeth, cartilage, and muscle. It can be benign or malignant.

Sebaceous Cyst (Wen, Epidermal Cyst)

It is a *retention cyst*. It is due to obstruction at the mouth of a sebaceous duct, causing a cystic swelling due to collection of its own secretion. It is common in face, scalp, and scrotum. **It is not seen in palms and plantar aspect of foot (sole)** as there are no sebaceous glands. Sebaceous cyst contains yellowish material with fat (sebum), epithelium (thick porridge like) which is having putty like consistency, with a parasite in the wall of the sebaceous cyst—**demodex folliculorum**. Its lining is only epidermal layer of squamous epithelium.

Clinical features: Painless swelling which is *smooth, soft, nontender, freely mobile, adherent to skin especially over the summit, fluctuant (positive Paget's test), nontransilluminating with punctum over the summit*. It moulds on finger indentation. **Punctum** is present over the summit in 70% of cases because here



Figs 3.52A and B: Implantation dermoid in different places of the body—finger, toe.



Fig. 3.53: Dermoid cyst ovary—on table look (Teratomatous dermoid of ovary).

sebaceous duct directly opens into the skin which gets blocked. Punctum is depressed black coloured spot over the summit of the sebaceous cyst. Because of the denuded squamous epithelium (keratin) it is black in colour. In 30% cases sebaceous duct opens into the hair follicle and so punctum is not seen. Sebaceous cysts often can be multiple commonly in face and scrotum. Often hairs are less or skin over the summit of the sebaceous cyst is bald (**Figs 3.54A to C**).

Complications: *Infection* and abscess formation; Surface may rupture and gets ulcerated with discharge and chronic inflammation, this discharge often spreads to surrounding tissues and hardens, and is called as—**Cock's peculiar tumour** which often resembles epithelioma (**Figs 3.56A and B**). It is a misnomer. Occasionally yellowish sebum discharges slowly through a wide punctum and becomes hardened, inspissated sebaceous material known as **sebaceous horn** (Length greater than its base diameter, is called



Figs 3.54A to C: Sebaceous cysts in face and scalp. Note the hair loss over the summit in sebaceous cyst. Punctum is clearly seen in sebaceous cyst face. Punctum is present in 70% of sebaceous cysts.

Sequestration dermoid – - Occurs in the line of fusion - Skin is not adherent (free) - Extends often into deeper plane or cavities through suture line - Punctum is absent - Bone resorption and indentation is common - Restricted mobility - Needs proper evaluation with X-ray / CT scan - Excision is done under general anaesthesia	Sebaceous cyst – - Occurs anywhere except palm and sole - Skin is adherent over summit - Subcutaneous plane – do not extend to deeper plane - Punctum is present – 70% cases - Freely mobile without bone resorption - Superficial swelling, mobile - Excision is done under local anaesthesia
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Figs 3.55A and B: Sebaceous horn in scalp and nape of neck.

as horn) (**Figs 3.55A and B**). Calcification also can occur in sebaceous cyst. Punctum is usually absent in sebaceous cysts of the scrotum (**Fig. 3.57**) (*Fordyce's disease* is heterotopic sebaceous glands in mucosa of lip and oral cavity).

Lipoma

It is a benign tumour arising from yellow fat (Tumour arising from brown fat is called as *hibernoma*). It is called as *universal tumour/ubiquitous tumour* as it can

Figs 3.56A and B: Cock's peculiar tumour over scalp and ear. It is a misnomer. It is not a tumour. It mimics epithelioma.

occur anywhere in the body (except in brain). It is the **commonest benign tumour**. It can be *diffuse or localised*. Diffuse lipomas are not encapsulated, not well localised. They are common in palm, sole, head and neck region, difficult to be removed. Diffuse type is often called as *pseudolipoma*. It is usually harmless except with some cosmetic problem. Lipoma can be single or multiple. Multiple lipomas (5%) are often associated with many syndromes like **MEN Syndrome** (*multiple endocrine neoplasia syndrome*). **Types** - Painful lipomas are called as *neurolipomas*. **Dercum's disease** is tender deposition of fat especially on the trunk, is also called as *adiposis dolorosa*. It is common in females. It is basically multiple *neurolipomatosis*. Fibrolipoma; naevolipoma; lipoma *arborigens* (pedunculated lipoma); neurolipoma are different



Fig. 3.57: Multiple sebaceous cysts in the scrotum. Punctum is usually absent in scrotal sebaceous cysts.

variants of lipoma. **Localised lipoma** is the commonest type. It is encapsulated type. It can occur anywhere but more commonly observed in nape of the neck, back, neck and shoulder. It is most common in subcutaneous plane. It also can be intermuscular; subfascial; intramuscular; parosteal; subserosal; submucosal; extradural; subdural (not intracerebral); subserosal; intraarticular or subsynovial. Lipomas attain large size in thigh, shoulder, retroperitoneum, back which may often turn into sarcoma (**Figs 3.58A and B**).

Clinical features: Localised painless swelling, which is lobular, nontender, semifluctuant (in normal body temperature fat is in semiliquid state and so often slight fluctuation may be elicited), freely mobile, with edge slipping between the palpating fingers (*slip sign*), with free skin. Using index finger edge of the lipoma when pushed will slip under the palpating finger. Naevolipoma shows dilated veins over the surface and so called as *lipoma telangiectasis*. Fibrolipoma contains more fibrous tissue and so it is firm. Neurolipoma also contains nerve tissue also and so is painful. At times lipomas may be pedunculated (**Figs 3.59A to C**). Lipoma is not transilluminant.

Differential diagnoses: Neurofibroma and other cystic swellings.

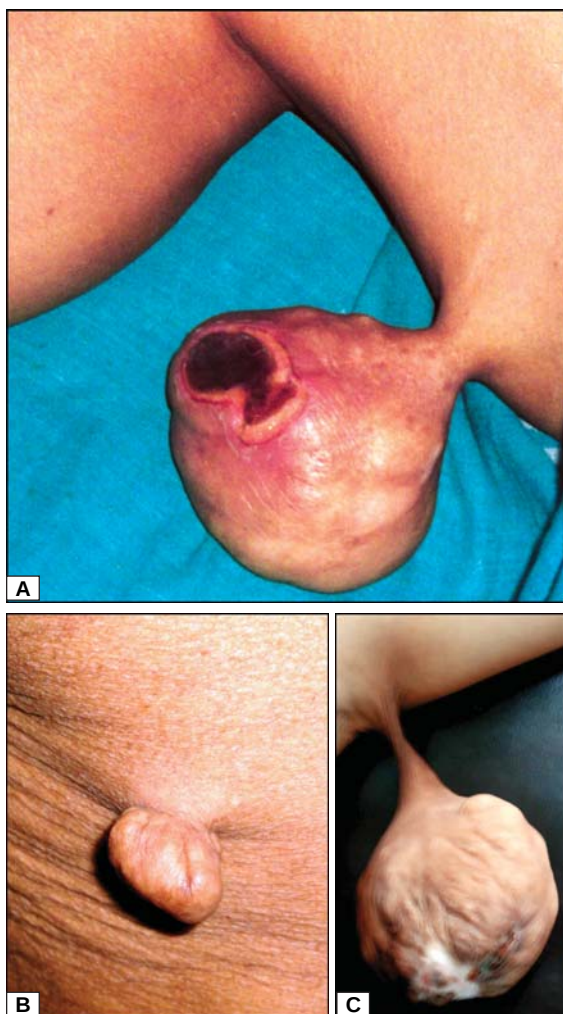


Figs 3.58A and B: Typical lipoma. Note the well defined, lobulated surface.

Complications: Sarcomatous change—liposarcoma; myxomatous change; saponification; calcification; submucosal lipoma in intestine can cause intussusception and so intestinal obstruction. Repeated trauma may cause ulceration over the summit which is more often seen in pedunculated lipoma.

Glomus Tumour

It is also called as *glomangioma*. It arises from the cutaneous glomus composed of a tortuous arteriole which communicates directly into the venule and these



Figs 3.59A to C: Pedunculated lipoma. Note the ulceration in one type. It is excised by elliptical incision.

vessels being surrounded by network of small nerves. It is special type of arteriovenous communication (*Sucquet-Hoyer canals*) with smooth muscle cells and glomus cells surrounding it. These glomus cells are either epitheloid or cuboidal cells. Both medullated and nonmedullated sensory nerves end in these glomus cells. They are often seen in limbs and common in nailbeds. They regulate the temperature of the skin. The tumour under the nailbed which is a common site is 2-3 mm in size. Tumour consists of a mixture of blood spaces, nerve tissue; muscle fibres derived from the wall of the arteriole, with large cuboidal

glomus cells—**angiomyoneuroma**. It does not turn into malignancy.

Clinical features: *Severe burning sensation and pain, out of proportionate to the size of the lesion.*

It is compressible and pain is more when the limb is exposed to sudden changes in temperature. Slightest pressure causes excruciating pain. It looks like a reddish blue spot which does not blanch on pressure. On increasing the pressure in the arm above systolic, pain disappears. Pyogenic granuloma and subungual melanoma are differential diagnosis but *typical pain* is diagnostic.

Neurofibroma

It is tumour arising from connective tissue (neural—ectodermal and fibrous—mesodermal) of the nerve. It can be single or multiple. Neurofibromas may be associated with pheochromocytomas, hypertension and few syndromes. Sites—**Cranial, spinal and peripheral.**

Types

- a. **Nodular neurofibroma** presents as single smooth, firm, tender (often) swelling which moves horizontally (perpendicular to the direction of the nerve), not along in the direction of the nerve. Pressure effects of the tumour over the nerve fibres cause pain, tingling sensation and hyperaesthesia/paraesthesia along the distribution of the nerve. Neurofibroma is the commonest intradural extra-medullary spinal tumour.
- b. **Plexiform neurofibroma** commonly occurs along the distribution of 5th cranial nerve in the skin of the face. It is more common in ophthalmic division of trigeminal nerve. It often occurs in the cutaneous distribution of the peripheral nerve. It attains enormous size with thickening of the skin which hangs downwards. It causes erosion into the bone, orbit and deeper structures. It may cause myxomatous degeneration also. It causes cosmetic problem. Rarely does it occur in upper limb. Development of sarcoma is very rare in this type. *Pachydermatocele* is a variant of plexiform neurofibromatosis observed in the neck (**Figs 3.60A and B**).
- c. **Generalised neurofibromatosis (Von Recklinghausen's disease):** It is an inherited autosomal dominant disease (congenital) wherein there will

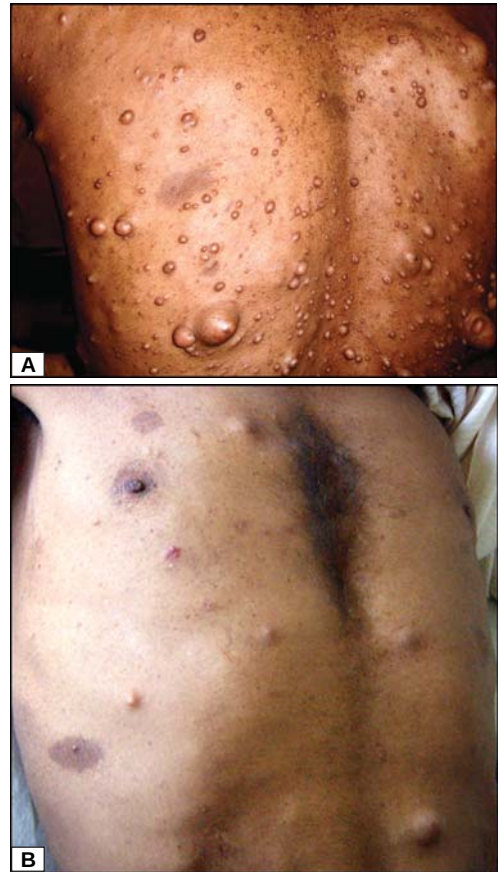


Figs 3.60A and B: Plexiform neurofibromatosis.

be multiple neurofibromas in the body (1:4000 live births; chromosome 17). It commonly involves peripheral nerves; often spinal and cranial nerves. So it is often classified as cranial, spinal or peripheral. It is commonly associated with pigmented spots (coffee coloured) in the skin, often seen on the back, abdomen, thigh (*café au lait spots*). It signifies common neuroectodermal origin of nerve sheath cells and melanocytes) (more than 5 in number with each more than 1.5 cm in size are significant) (**Figs 3.61A and B**). Axillary or groin freckles with *Lisch nodules* may be present. Familial neurofibroma may be associated with scoliosis or MEN II b syndrome (Multiple Endocrine Neoplasia syndrome type II b-medullary carcinoma of thyroid; pheochromocytoma; hyperparathyroidism; multiple neurofibromas in eyelids, lips and face) (**Figs 3.62 to 3.64**).

d. Elephantiac neurofibromatosis: It is of congenital origin involving limbs. Skin of the limb is greatly thickened, dry and coarse.

Complications: *Sarcomatous changes* (5%): When it occurs it shows rapid enlargement, warmth, more vascularity with dilated veins. Persistent severe pain; fixity and fungation also can occur. Secondaries in lungs can occur through blood spread. *Haemorrhage* can occur into the tissues. Spinal and cranial neurofibromas can cause neurological deficits. *Erosion* can occur into the deeper planes, bone, orbit. *Calcifications*, *saponification*, *myxomatous changes* and



Figs 3.61A and B: Café au lait spots in the back. It is a feature of von Recklinghausen's disease of neurofibromatosis. It is coffee coloured pigment spots in the skin. More than 5 in number with each more than 1.5 cm in size is significant.



Figs 3.62A and B: Multiple neurofibromas (von Recklinghausen's disease).



Fig. 3.63: Multiple neurofibromas with intestinal neurofibroma. It may precipitate intussusception.



Fig. 3.64: Neurofibrosarcoma in a female with multiple neurofibromatosis.

pressure symptoms are other complications. Intestinal neurofibroma may precipitate *intussusception*.

Neurilemmoma (Schwannoma)

It is arising from ectodermal Schwann (neurilemmal cells) cells. They are benign, lobulated, encapsulated, soft, and whitish in appearance. They displace the nerve from which they arise and can be removed. *Anthony A type* contains two rows of spindle cells with central acellular Verocay bodies. *Anthony B type* contains acellular amyloid areas. They are common in acoustic

nerve but do can occur in a peripheral nerve. Occasionally they are multiple. Present as freely mobile swelling; with pain along the distribution of the nerve, hyperaesthesia, and tenderness. *Recurrent schwannoma could be malignant (Malignant schwannoma are very aggressive).*

Neuroma

Two types of neuromas are found—**False neuroma and true neuroma.**

False neuroma: It occurs due to injury to the nerve either after trauma or amputation which presents as tender swelling. It arises from the connective tissue of nerve sheath. It contains fibrous tissue with coiled nerve fibres. It can be *end neuroma* commonly seen in amputation stump as a tender localised firm swelling often adherent to the stump scar. It can cause neuralgia in the stump. It prevents proper usage of the prosthesis. So during amputation, nerve should be pulled down and cut so as to make cut end to retract more proximally which prevents it to form neuroma. It can be *lateral (side) neuroma* which is observed after partial nerve injury usually after trauma. It presents as a tender, firm swelling along the line of the peripheral nerve (**Fig. 3.65**).

True neuroma: It is rare tumour. It occurs in connection with sympathetic system.

Types:

Ganglioneuroma—It contains ganglion cells and nerve fibres. It occurs in connection with sympathetic chain. So it is observed in neck, thorax, retroperitoneum,

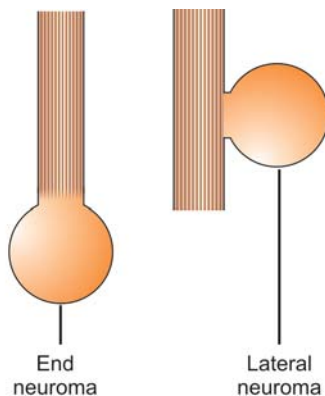


Fig. 3.65: Types of false neuroma—end and lateral.

adrenal medulla. It is relatively benign symptomless and often attains large size.

Neuroblastoma—It is poorly differentiated, aggressive, embryonic type of tumour. It is seen in infants and children. It spreads through blood but can go for spontaneous remission occasionally.

Myelinic neuroma: It contains only nerve fibres. Here ganglion cells are absent. It occurs in spinal cord or pia mater.

Ganglion

It is a cystic swelling occurring in relation to tendon sheath or synovial sheath or joint capsule. It contains clear gel-like fluid.

Common sites are: Dorsum of the wrist (near scaphoid-lunate articulation); flexor aspect of wrist around ankle joint—occasionally.

Pathogenesis: Possibilities thought of are—cystic degeneration of the tendon sheath; leakage of synovial fluid through joint capsule; presence of small islets of microspaces in synovial sheath which often fuse together or one of them gets enlarged to form ganglion.

Clinical features—Well-localised, smooth, soft, cystic, or tensely cystic (Paget's test is positive), nontender, transilluminant, swelling which is mobile but mobility is restricted when tendon is contracted against resistance (**Figs 3.66A to D**). Occasionally it communicates with joint capsule. Often pain, tenderness and restricted joint movement may be the presentation (but rare).

Differential diagnosis—Lipoma; lymph cyst; sebaceous cyst; neurofibroma. Patient should be explained of *high recurrence rate* (30%) after excision.

Papilloma

It is warty swelling from the skin or often from the mucous membrane (**Fig. 3.67**). It has got a central axis of connective tissue, blood vessels and lymphatics. *True papilloma*—It is a benign tumour with localised overgrowth of the all layers of the skin. It contains sweat glands, sebaceous glands and hair follicles. It is commonly pedunculated but rarely can be sessile. *Pedunculated papilloma* is villous with a central axis



Figs 3.66A to D: Ganglion over the wrist. Its mobility should be checked both with wrist relaxed as well as with wrist extending against resistance. Skin should be held/pinched to confirm that ganglion is not fixed to skin.

of connective tissues, blood vessels and lymphatics (**Figs 3.68 and 3.69**). *Infective papilloma* is a warty lesion due to infection. For example, *Condyloma acuminata*. *Papilloma* may be single or multiple. *Papilloma* may be pigmented or nonpigmented. *True papilloma* may turn into *squamous cell carcinoma* occasionally. There will be sudden increase in size, bleeding or ulceration. Mechanical disability

like voice change is observed when it occurs in vocal cord.

Differential diagnosis: Amelanotic melanoma, pedunculated lipoma, carcinoma. Papilloma can occur in the breast called as *duct papilloma* which is the commonest cause of bloody discharge from the nipple. Papilloma can occur in mucous membrane like in *oral cavity*, *urinary bladder* (transitional papilloma), in the



Fig. 3.67: Papilloma tongue. Note: Papilloma can also occur in mucous membrane like in oral cavity, urinary and gallbladder, rectum.

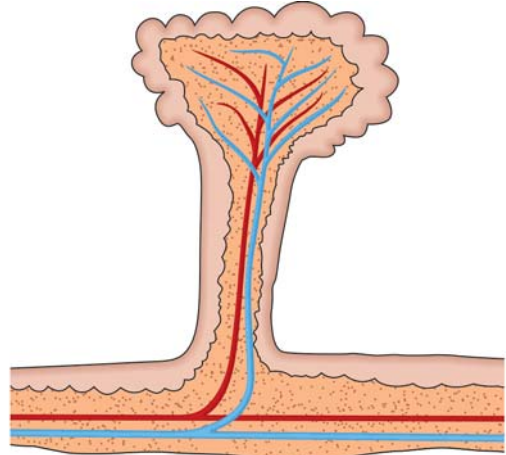


Fig. 3.68: Pedunculated papilloma with its pedicle.

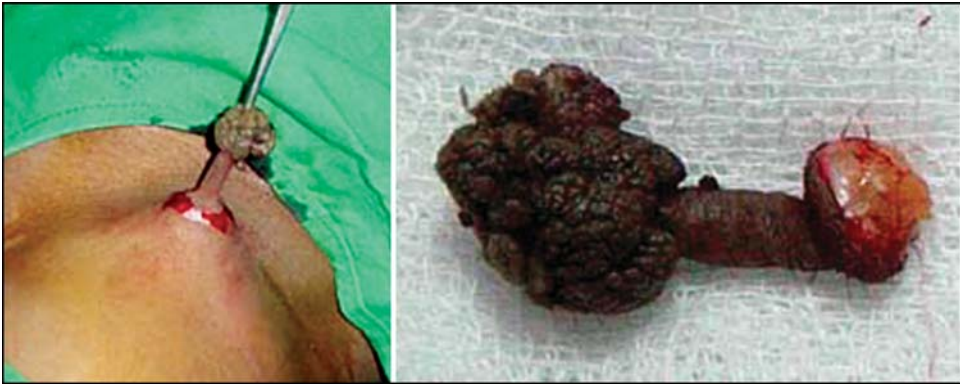


Fig. 3.69: Papilloma which is pedunculated. Note its base.

rectum (columnar), in the *larynx*, in the *gallbladder* (cuboidal) (**Figs 3.67 and 3.70**).

Fibroma

It is a benign tumour arising from fibrous tissue. It is capsulated.

Classification of true fibroma:

1. *Soft fibroma*: Contains immature fibrous tissue. Common in face, presents as soft brown swelling.
2. *Hard fibroma*: Contains well-formed fibrous tissue. True fibroma is rare and cannot be diagnosed clinically. It is mostly combined with mesodermal tissues like nerve sheath (neurofibroma), fat (fibrolipoma), muscle (fibromyoma). An entity called



Fig. 3.70: It is often called as condyloma. It is dry, raised lesion in the anal canal. Condyloma lata is seen in secondary syphilis. Condyloma acuminata is of viral origin.

aggressive fibromatosis is known to occur as unencapsulated proliferation of fibrous tissue, common in abdominal and, chest wall. It is presently considered as *locally malignant*. It does not spread through lymphatics or through blood. *But recurrence is common*. **Desmoid tumour** is a variant of aggressive fibromatosis, seen in females, often associated with Gardner's syndrome. (Desmos = tendon, eidos = appearance). **Recurrent Fibroid of Paget's** is a rare type of fibrosarcoma occurring in a scar tissue after many years.

Bursae

Bursa is a sac like cavity containing fluid within, which in normal location prevents friction between tendon and bone. It is smooth, soft/firm (tensely cystic), fluctuant, cystic. Skin may be free or often adherent due to chronic inflammation. Sometimes tenderness can occur in bursae due to acute inflammation or abscess formation. Usually it is mobile but inflammation may restrict the mobility. It should be differentiated from sebaceous cyst, soft tissue tumour, lipoma, neurofibroma. Minor injuries and pressure leads into bursitis, which will present as a swelling at the site. Inflammation of this bursa due to friction causes **bursitis**, which commonly presents as swelling, pain, and restricted movements.

Different types: It can be *anatomical or adventitious*.

Anatomical

Subhyoid bursa: A horizontally oval swelling situated below the hyoid bone and in front of the thyrohyoid membrane.

Subacromial bursa: In front and lateral to humeral head in relation to supraspinatus tendon between acromion and greater tuberosity of humerus. **Bicipito radial bursa.** **Olecranon bursa** (*Student's elbow*, *Miner's elbow*) (**Figs 3.71 to 3.73 and 3.75**).

Psoas bursa: A tensely cystic swelling beneath and below the inguinal ligament on the lateral aspect of the femoral triangle. But it will not extend above the inguinal ligament into the iliac region (unlike in psoas abscess which extends above and is cross fluctuant).

Prepatellar bursitis (*Housemaid's knee*): It occurs in front of lower part of patella and upper part of patellar



Fig. 3.71: Bursa near elbow joint.



Fig. 3.72: Bursa over elbow joint.



Fig. 3.73: Adventitious bursa over lateral aspect of foot—a common site.

tendon due to constant pressure (like kneeling) (**Fig. 3.74**).

Infrapatellar bursitis (*Clergyman's knee*): It is inflammation of bursa occurring in relation to lower half of the patellar tendon (**Fig. 3.74**).

Semimembranosus bursa; Bursa anserina: Located under the tendons of Guy ropes (sartorius, gracilis and semitendinosus tendons) (*Goose's foot*).

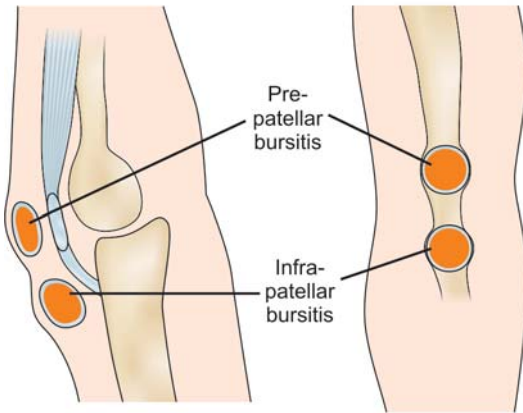


Fig. 3.74: Prepatellar (Housemaid's knee) and infrapatellar bursae (Clergyman's knee).



Fig. 3.76: Common site of adventitious bursa (Bunion).

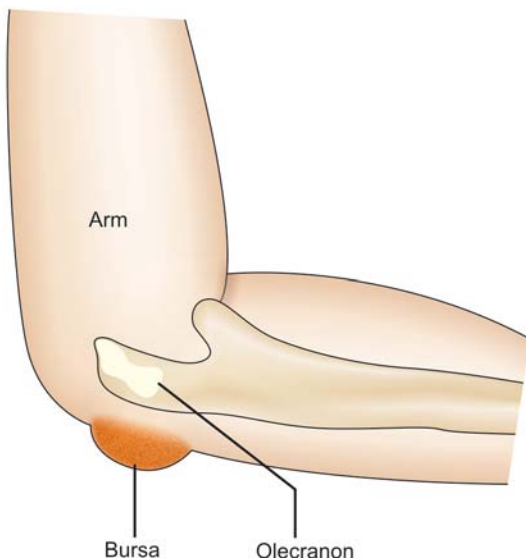


Fig. 3.75: Location of olecranon bursa (Student's elbow).

Retrocalcaneum bursitis—occurs between calcaneum and tendoAchilles.

Adventitious bursa occurs in an unusual site like in hallux valgus (*bunion*) over first metatarsal, over lateral malleolus (*tailor's bursa*), between clavicle and skin near shoulder (*porter's bursa*), between gluteus maximus muscle and ischial tuberosity (*weaver's bursa*), between tendoachilles and skin (*retroachilles bursitis*) or over gluteal tuberosity (**Fig. 3.76**). It occurs due to friction or pressure.

Semimembranosus Bursa

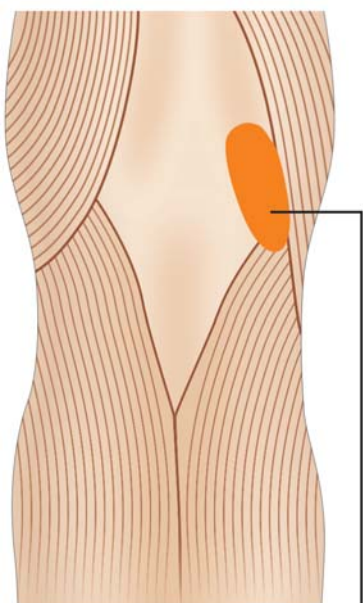
It is a cystic swelling in the upper medial aspect of the popliteal fossa under the semimembranosus tendon. It is said that friction under the tendon causing bursitis.

Clinical features: It is common in young individuals. It is soft, smooth, cystic, often transilluminant and nontender, noncompressible swelling located in upper and medial aspect of the popliteal fossa. On flexion of knee the swelling disappears and on extension it becomes more prominent. Swelling does not communicate into the knee joint cavity. Here knee joint is normal (**Figs 3.77 and 3.78A and B**).

Morrant Baker's Cyst

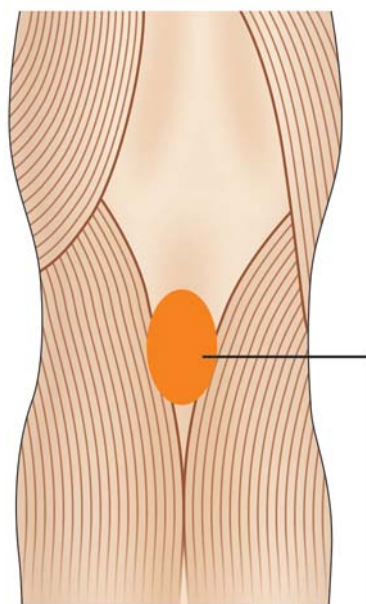
It is a cystic swelling containing gel-like fluid in the lower midline of the popliteal fossa. It occurs due to herniation of the synovial membrane of the knee joint as a result of chronic arthritis.

Clinical features: It is common in middle-aged individuals. It is smooth, soft and cystic, often tender swelling located below and on midline of the popliteal fossa (**Fig. 3.79**). On flexion swelling increases and on extension swelling decreases in size. Pain and tenderness are present in knee joint with effusion showing positive patellar tap. The knee joint movements are painful and restricted.



SM bursa—medial

Fig. 3.77: Semimembranosus bursa—typical location.



Baker's cyst below midline

Fig. 3.79: Marrant-Baker's cyst—typical location.



Fig. 3.78: Semimembranosus bursa—typical location. On flexion it disappears.

Lymph Cyst (Lymphatic Cyst)

It is an *acquired type of distension cyst* wherein lymphatics form a localised swelling with a capsule around it. This localised cystic swelling contains lymph. It is commonly due to trauma. Trauma causes disruption of subcutaneous lymphatics causing accumulation of lymph in a localised area with a capsule. It usually occurs in subcutaneous plane, which is well-defined, smooth, soft, nontender, mobile, non-compressible, fluctuant (*positive Paget's test*), and brilliantly transilluminant (**Fig. 3.80**). It is usually not adherent to the overlying skin. **Common sites** are in limbs and neck. It can get infected and form an abscess.

Differential diagnosis: Cold abscess, dermoid cyst.

Calcinosis Cutis

It is a type of calcification (dystrophic) in or under the skin. It usually presents as a circumscribed lesion in the skin. It is commonly seen in females and common site is in the waist (**Fig. 3.81**). It is usually bilateral. It is said to be due to friction causing degeneration of skin and immediate deeper structure with increased



Fig. 3.80: Lymph cyst, which is transilluminant. It is an acquired condition.



Fig. 3.81: Calcinosis cutis near waist is a common site. It is common in females.

local alkalinity of the tissue causing precipitation of the calcium leading to solid, hard, swelling in the skin. Cut section shows hard, yellowish material. It may mimic calcified lipoma or neurofibroma.

Callosity and Corn

They are thickened, hyperkeratotic skin due to pressure and repetitive minor trauma. They are common in old people due to old age skeletal changes causing maldistributed weight bearing.

Callosity is thickened, grayish brown hyperkeratotic patch of skin commonly of hands and feet over an area of excessive wear and tear in relation to occupation. It protrudes outwards. When top layer is removed, shiny, translucent, homogenous dead skin layer can be exposed. It is *not a painful* condition. It can be left alone (**Fig. 3.82**).



Fig. 3.82: Callosity foot. It is outward protruding grayish brown hyperkeratotic patch of skin in the foot/hand. It is not a painful condition.

Corn is a localised, smaller, deeper lesion with palpable tender nodule having a yellow white core of cornified dead skin in the centre. It is common in soles, tips of toes. Often corns are multiple and bilateral (**Fig. 3.83**). It is often due to friction like using ill-fitting footwear. Corns are *painful and tender*. Corn can be commonly hard in the sole or rarely soft corn do can



Fig. 3.83: Corn foot. It is localised, painful, tender, deep lesion with a deep core that contains degenerated dead keratotic cells and cholesterol.

occur between 4th and 5th toes due to friction of bases of adjacent proximal phalanges. Corn consists of severe keratoses with central degenerated dead cells and cholesterol. Corn should be differentiated from plantar wart by removing the top layer of the lesion so as to expose dead deep core of corn or soft branching process of wart.

Chordoma

It is tumour arising from the remnants of notochord. It is seen in sacrococcygeal region; sphenoidal sinus region or around the foramen magnum. It invades into surrounding structures like nerves. Often it is aggressive (**Fig. 3.84**).



Fig. 3.84: Chordoma over the sacrum.

Cellulitis

It is *spreading inflammation of subcutaneous and fascial planes*. Infection may follow a small scratch or wound or incision. Common causative agents are *Streptococcus pyogenes* organisms and other gram positive organisms. Often gram negative organisms like *Klebsiella*, *Pseudomonas*, *E. coli* are also involved. Cellulitis can be superficial or deep. **Sequelae of cellulites** - Infection can get localised to form **pyogenic abscess**; Infection can spread to cause **bacteraemia**, **septicaemia**, **pyaemia**; Often infection can lead to **local gangrene**.

Clinical features are fever, toxicity (tachycardia, hypotension); diffuse swelling which is spreading in nature; pain and tenderness, red, shiny, boggy area with stretched warm skin. Brawny look of the area

with pitting oedema without any edge are the typical features (Absence of edge; absence of fluctuation; absence of pus; absence of limit). Cellulitis will progress rapidly in diabetic and immunosuppressed individuals like patients with HIV infection. Associated lymphangitis is often seen as raised red streaks which blanch on pressure. Tender palpable regional lymph nodes are common due to associated lymphadenitis. Often these lymph nodes get suppurated forming an abscess eventually (**Figs 3.85 to 3.87**).

Orbital cellulitis: Cellulitis in orbit causes proptosis, leading to impairment of ocular movements and blindness. It can spread through ophthalmic veins into cavernous sinus causing **cavernous sinus thrombosis**. It requires admission and immediate aggressive treatment with higher generation antibiotics.



Fig. 3.85: Cellulitis left leg with redness, oedema.



Fig. 3.86: Cellulitis face.



Fig. 3.87: Extensive cellulitis of arm and forearm with abscess formation.

Ludwig's angina: It is cellulitis of upper part of the neck involving submandibular region and floor of the mouth along the fascial planes.

Clinical features: Diffuse swelling, redness, tenderness and induration in the floor of the mouth and submandibular region; difficulty in opening the mouth (*Trismus*); toxic features like fever, tachycardia and tachypnoea; **severe laryngeal oedema** (presents with respiratory distress, stridor and cyanosis).

Complications—Septicaemia; spread of infection into the parapharyngeal space leads to *thrombosis* of internal jugular vein which may extend above into the sigmoid sinus which may be fatal.

Erysipelas: It is a spreading inflammation of the skin and subcutaneous tissues due to infection caused by *Streptococcus pyogenes*. There will be always cutaneous lymphangitis with development of rose pink rash with cutaneous lymphatic oedema. Vesicles which form eventually will rupture to cause serous discharge. Common sites are—orbit, face and scrotum. In face and orbit it causes severe oedema.

Clinical features are toxaemia; rash which is fast spreading and blanches on pressure; rash is raised with sharp margin; discharge is serous (In cellulitis discharge is purulent); *Milian's ear sign* is a clinical sign used to differentiate erysipelas from cellulitis wherein ear lobule is spared. Skin of ear lobule is adherent to the subcutaneous tissue and so cellulitis cannot occur. Erysipelas being a cutaneous condition can spread into the ear lobule. Disease is common in poor hygienic debilitated individuals.

Erysipeloid disease is also called as 'Fish handler's disease'. It is a self limiting disease with mild features of both cellulitis and erysipelas. It occurs following minor trauma in fish and meat handlers. It is common in hands.

Abscess

Pyogenic Abscess

It is a **localised collection** of pus in a cavity lined by granulation tissue, covered by pyogenic membrane. It contains pus in loculi. Pus contains dead WBCs, multiplying bacteria, toxins and necrotic material. Spread may be direct, haematogenous, lymphatics from adjacent tissues. *Staphylococcus aureus* and *Streptococcus pyogenes* are common organisms. It is often an effect of cellulitis or lymphangitis. Abscess is more common in malnourished people, people with anaemia, diabetes mellitus, HIV, immunosuppression or old age. Trauma, haematoma, virulence of the organisms are other factors.

Clinical features: Throbbing pain; fever with chills and rigors; soft, smooth, tender, fluctuant swelling with often visible pus and pointing tenderness. Brawny induration is common in surrounding area. Redness, warmth with restricted movements of the part or adjacent joint are observed (**Figs 3.88A and B**). *Visible (pointing) pus, tenderness, fluctuation* are the features of *formed abscess* (Commonly cellulitis occurs first which eventually gets localised to form an abscess).

Sites of an abscess: It can be *external or internal* depends on whether the abscess is on the surface or in the deeper cavities like abdomen. **Examples of external sites** are: fingers and hand; neck; axilla; breast; foot; thigh—here it is deeply situated with brawny induration; ischiorectal and perianal region; abdominal wall; dental abscess; tonsillar abscess and other abscesses in the oral cavity. **Examples of internal abscess** are: abdominal: subphrenic, pelvic, paracolic, amoebic liver abscess, pyogenic abscess of liver, splenic abscess, pancreatic abscess; perinephrenic abscess; retroperitoneal abscess; lung abscess; brain abscess.

Complications of an abscess: Bacteraemia, septicaemia, and pyaemia; multiple abscess formation; metastatic abscess; destruction of tissues due to necrosis;



Figs 3.88A and B: Abscess in the groin. Redness, pain, localised swelling, warmth, fluctuation, often visible pus, brawny induration are the features of an abscess. Abscess should be aspirated prior to surgical drainage to rule out differential diagnosis like aneurysm.

antibioma formation due to antibiotic therapy without drainage (common in breast abscess); sinus and fistula formation; large abscess may erode into adjacent vessels and can cause life threatening torrential haemorrhage (examples: pancreatic abscess causing splenic vessel haemorrhage, psoas abscess causing iliac vessel haemorrhage); abscess in head and neck region can cause laryngeal oedema, stridor and dysphagia. *Specific complications of internal abscess:* Brain abscess can cause intracranial hypertension, epilepsy, neurological deficit; liver abscess can cause hepatic failure, rupture, jaundice; lung abscess can lead on to bronchopleural fistula or septicaemia or respiratory failure or ARDS. **Abscess should be formed before draining.** *Exceptions for this rule are:* Parotid abscess; breast abscess; axillary abscess; thigh abscess; ischio-rectal abscess. **Differential diagnoses to be remembered before draining an abscess are—***Aneurysm* especially in popliteal, femoral and axillary



Fig. 3.89: Abscess in forehead region in a diabetic patient.

regions (**Fig. 3.89**). So using a needle always aspirate and confirm the pus; **Soft tissue tumours**—Sarcomas may be smooth, soft/ firm and warm with dilated vessels on the surface.

Cold Abscess

It means there are no signs of acute inflammation like redness, warmth, tenderness. It is painless, smooth, soft, fluctuant, nontransilluminating. Oedema, brawny indurations are absent. Cold abscess is due to caseative necrosis of tuberculous disease. It is commonly observed in caseating tuberculous lymphadenitis; tuberculosis of spine; joint tuberculosis; tuberculosis of ribs, mediastinum, etc. Cold abscess can occur at the site of the disease like in the neck (neck is the commonest site) (**Fig. 3.90**) or often caseating fluid



Fig. 3.90: Cold abscess sternum. Neck is the common site.

can travel along the fascial or neurovascular bundle to cause abscess at different sites. Such cold abscess is frequently observed in tuberculosis of spine (T_{10}). Psoas abscess; groin abscess; abscess in paraspinal region; abscess in intercostal space are the examples of such type of cold abscess.

Difference between pyogenic and cold abscess	
Pyogenic abscess	Cold abscess
Red, warm, tender, with signs of acute inflammation	No signs of acute inflammation
Pyogenic bacteria are nonspecific organisms (Streptococci / Staphylococci)	Tuberculous bacteria
For drainage, dependent incision is used	Nondependent incision is used
Suturing of the wound is not done	Cavity is curetted and sutured
Drain is placed	Drain is not placed

Pyæmic Abscess

These are formation of multiple abscesses in the different parts of the body like subfascial plane, deeper planes, in the organs like liver, lungs, brain, spleen, etc. It is due to lodging of the multiple infective bacterial emboli from the circulating blood at different places which cause suppuration and abscesses formation (**Fig. 3.91**). *Subfascial pyæmic abscesses* often do not show the features of acute abscess like warmth, fluctuation, pointing tenderness.

Boil (Furuncle)

It is an acute *Staphylococcus aureus* infection of a hair follicle with perifolliculitis which usually proceeds to suppuration and central necrosis. It is common in neck, back and upper limb. Often boil opens on its own and subsides. Furuncle in external auditory meatus is very painful because of rich cutaneous nerves and firmly adherent skin to perichondrium. Pain, indurated swelling, greenish pustule that eventually rupture to create a deep cavity with green slough, often with tender palpable regional nodes are the features (**Fig. 3.92**). Once it ruptures red granulation tissue forms in the surface/floor and spontaneous healing takes place with antibiotic coverage.

Complications: Cellulitis; lymphadenitis; hydradenitis (in axilla—infection of group of hair follicles).

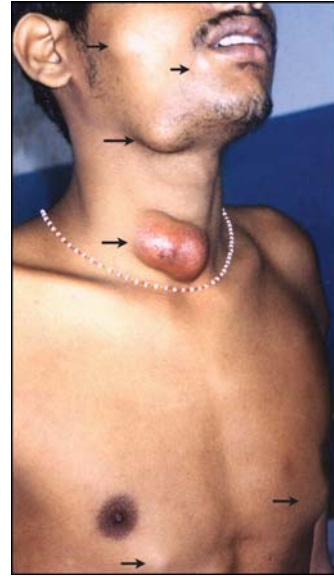


Fig. 3.91: Multiple abscesses. It could be due to septicaemia (pyaemia), immunosuppression or diabetes mellitus.



Fig. 3.92: Furuncle/boil—infection of hair follicle.

Hidradenitis Suppurativa

It is a chronic infective and fibrous disease of the skin which bears apocrine sweat glands. Apocrine sweat glands are coiled sweat glands which open into hair follicle. It is common in axilla, areola, umbilicus, groin and perineum. In the axilla condition is often bilateral. It is related to obesity, smoking, poor hygiene, diabetes mellitus, steroids. Common bacteria are staphylococci, streptococci and propionibacterium acnes. Keratin

blocks the duct of the apocrine sweat glands causing dilatation of the duct leading into infection and suppuration of the glands. Many adjacent glands involve eventually causing fibrosis, scarring and sinus formation. Commonest site is *axilla* (**Fig. 3.93**). It is common in *females* (4:1). Discharging sinuses, induration, tenderness and oedema are common. It often looks like tuberculosis or malignancy.



Fig. 3.93: Hidradenitis suppurativa—axilla.

Carbuncle

Word meaning carbuncle is charcoal. It is an infective gangrene of skin and subcutaneous tissue. *Staphylococcus aureus* is the main causative organism. Common site of occurrence is *back and nape of neck* (**Fig. 3.94**). It is common in *diabetics* and after forty years age. It is common in males. Infection → red, indurated oedematous area → small vesicles develop → discharge through multiple openings → sieve-like pattern/cribriform pattern → many fuse together to form a central necrotic ulcer with peripheral fresh vesicle looking like a ‘*rosette*’ with ash gray slough → skin becoming black due to blockage of cutaneous vessels → disease spreads to adjacent skin rapidly. Patient will be **toxic** and in diabetic they are **ketotic**.

Renal carbuncle is an entity which occurs in kidney due to infection, forming localised infective mass lesion.



Fig. 3.94: Carbuncle in the nape of neck. It is an infective gangrene of skin and subcutaneous tissue.

Pott's Puffy Tumour

It is formation of diffuse external swelling in the scalp due to *subperiosteal pus formation and scalp oedema*. It originates commonly in frontal region and may extend into other regions. It is usually due to chronic frontal sinusitis which eventually suppurates and extends into subperiosteal region but trauma also can cause the same.

Clinical features: Pain and diffuse swelling in frontal region which is warm, tender. Swelling often extends to face and eyelids (**Fig. 3.95**). Patient will be toxic and drowsy.

Complications: *Osteomyelitis* of frontal bone; spread of infection into intracranial cavity leading to *intracranial abscess* (Extradural or subdural abscess). So it may present with features of raised intracranial tension like headache, coning and convulsions.

Pyogenic Granuloma (Granuloma Pyogenicum)

It is a common condition which occurs in face, scalp, nose, fingers and toes (**Figs 3.96A and B**). It may be due to minor trauma or minor infection. Infection leads to formation of unhealthy granulation tissue which protrudes through the wound.



Fig. 3.95: Pott's puffy tumour.

Clinical features: Usually single, well localized, red, firm, nodule, which bleeds on touch. It is rapidly growing relatively painless and often mimics haemangioma, papilloma, skin adnexal tumour, squamous cell carcinoma and melanoma.

Bacteraemia, Septicaemia, Pyaemia

These conditions are discussed here as they may cause multiple abscesses in the body or these conditions may occur due to existing abscess itself. *Bacteraemia* is presence of bacteria in blood. It causes fever with chills and rigors, tachycardia and leucocytosis. It may get controlled by antibiotics or may lead into septicaemia (septic shock). *Septicaemia* is presence of overwhelming, multiplying bacteria in the blood with toxins causing *systemic inflammatory response syndrome (SIRS)* or *multiorgan dysfunction syndrome (MODS)*. Patient presents with fever, oliguria, jaundice, hypotension, feeble pulse, respiratory failure and drowsiness. Fever often may be absent or hypothermia may be evident due to severe sepsis wherein pyogenic response is absent. Septicaemia may be due to gram positive or gram negative organism. Gram positive septicaemia is due to staphylococci, streptococci, pneumococci, etc. *Overwhelming Postsplenectomy Infection (OPSI)* is a classical example of gram positive septicaemia. Gram negative septicaemia is commonly observed in urinary infection, biliary sepsis, peritonitis,



Figs 3.96A and B: Pyogenic granuloma post-auricular region and nose.

abdominal infection, sepsis in diabetics and immuno-suppressed. It is also called as *endotoxic shock* due to endotoxins released from lysed bacteria. Common gram negative organisms causing gram negative septicaemia are *E. coli*, *Klebsiella*, *Pseudomonas* and *Proteus*. Initial warm reversible stage when becomes severe forms eventually irreversible cold stage which is often difficult to manage. It is always better if septicaemia is diagnosed in initial warm stage itself. Pyaemia is presence of multiplying bacteria in blood

as emboli which spreads and lodges in different organs in the body like liver, lungs, kidneys, spleen, brain causing **metastatic abscess**. This may lead to **Multi Organ Dysfunction Syndrome (MODS)**. It may endanger life if not treated properly.

Seborrhoeic Keratosis (Seborrhoeic Wart, Basal Cell Papilloma)

It is a benign overgrowth of the basal layer of epidermis with excess of small darkly stained basal cells, which protrudes from the surface of the epidermis to give oily appearance.

Features: It is common in elderly. It is common in Caucasians. It is often familial with autosomal dominant gene transmission. Common sites are the back, face and neck. It grows slowly with widening in area without altering in thickness. It often gets infected but uncommonly bleed on touch. It is pigmented due to melanin and so mimics naevus or melanoma or pigmented BCC. Often when it scabs off it leaves a pale pink patch on the skin with visible small surface capillaries. It is *not a premalignant condition* (Note: Solar keratosis is a premalignant condition). It is hard and stiffer than normal skin. Lymph nodes are not involved. It does not occur in palms and soles. It can be picked off from the skin. 'Stuck on' appearance is characteristic (**Fig. 3.97**).



Fig. 3.97: Seborrhoeic keratosis in face. It is not a premalignant condition.

Senile or Solar Keratosis

It is multiple, hyperkeratotic, dry, scaly/patchy, yellowish gray/brown lesion in the sun exposed parts of the skin like face, rim of the ears, dorsum of hands and fingers often forming an ulcer with a raised edge. It is often seen in elderly men who were working outdoors for many years. It is a benign lesion confined to skin but with malignant potential. Squamous cell carcinoma can occur in a long standing solar keratosis after ten years or more. Fixity or tethering, everted edge, recent increase in size and nodal spread may be the features of malignant transformation.

Impetigo

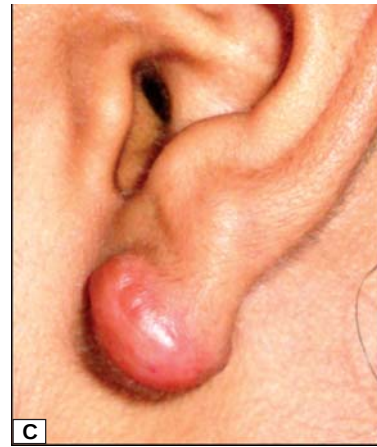
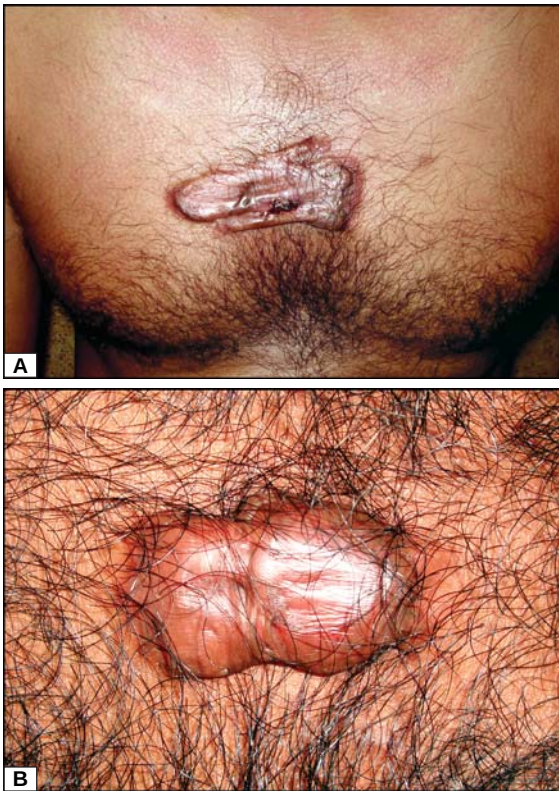
It is highly infectious superficial skin infection caused by staphylococci/streptococci organisms. It is usually seen in children, with formation of multiple blisters that rupture and coalesce, to be covered with honey coloured crust. **Scrumptox** is a type of impetigo seen in Rugby players due to staphylococcal infection.

Keloid

Word meaning keloid is 'like a claw'. It is abnormal proliferation of immature fibroblasts, immature blood vessels and type III *thick* collagen stroma. There is defect in maturation and stabilisation of collagen fibrils. It is common in blacks (15 times), females, Negroes. It is often familial. It may be associated with *Ehlers-Danlos syndrome* or scleroderma. It is common over sternum, upper arm (BCG vaccination scar), upper chest wall, ear and lower neck (**Figs 3.98A to D**). Scar of minor injury also can form keloid. Fibrous tissue continues to grow even after 6 months to many years. It extends like finger into adjacent normal skin and attains *vascularity*. It forms *pinkish black, painful, hyperaesthetic, tender swelling* (not a tumour but tumour like) which spreads and causes itching. It can occur as *spontaneous keloid* without a scar after an unnoticed trauma which is common in *Negroes*. Recurrence is common if excised.

Hypertrophic Scar

It is overgrowth of fibrous tissue (type III *fine* collagen) in any scar which is *limited to scar area only*; which grows upto 6 months; *not* genetically predisposed (unlike keloid); will *never* extend to normal adjacent



Figs 3.98A to D: Keloid over the chest (sternum), ear lobule (ear prick site) and shoulder.

skin; occurs *anywhere in the body*; self limiting; *not vascular*. It is common on the *flexor* aspect. It is *equal* in both sexes. There is *no* racial discrimination. *Precipitating factors* are lacerated wounds, infected wounds, scars healed by secondary intention, burns wound, scars which cross the Langer's line (**Fig. 3.99**).



Fig. 3.99: Hypertrophic scar in the thigh.

Warts

They are usually multiple hyperkeratotic skin patches with finger like projections, common in children and

adolescents. They are common in fingertips, face, axilla and sole of the feet. It may be familial but often stimulated by virus. They are dry, overgrown projections from the skin of finger often painful, tender and disfiguring. Repeated rubbing may cause infection. It can spread to other fingers and other parts of the body. Kiss lesions can occur. **Plantar wart (*Verruca plantaris*)** is wart in the sole. Specialty of this is it gets pushed into the sole of the foot. It is common in ball and heel of the foot. It is pearly white in colour with brownish flecks. It is often covered by apparently normal skin because wart is buried into the skin. It looks like a circular pit. It is gray white finger/filiform like strands in the centre of the lesion and is soft. Plantar warts can be multiple. It is painful and very tender on pressing (more than callosity or corns).

Keratoacanthoma

It is also called as *Molluscum sebaceum*. It is an overgrowth and subsequent spontaneous regression

of the sebaceous gland which opens into hair follicle often seen in adults (males 3:1). It is *self limiting benign neoplasm* probably of papilloma virus origin. It is proliferating squamous cells of the sebaceous glands which protrude out through the sebaceous duct. This solitary lesion is more common in the skin where more sebaceous glands are present. It is a painless rapidly growing hard mobile swelling of the skin (grows upto 4-8 weeks) with a central brown area which over 4-6 months leads into *spontaneous regression*. Central brown area is hard, peripheral rim is firm and rubbery (*volcano like*). Central area separates from lesion leaving a deep scar. Regional lymph nodes are not enlarged. It can be recurrent in lips and fingers. It *mimics* squamous cell carcinoma (epithelioma). It is a *pseudomalignancy* (**Fig. 3.100**).



Fig. 3.100: Keratoacanthoma. Note the central brown area. It mimics epithelioma.

Rhinophyma (Potato Nose, Bottle Nose)

It is a glandular form of acne rosacea causing immense thickening of distal part of the skin of nose with visible openings of sebaceous follicles. It is due to hypertrophy and adenomatous changes in sebaceous glands. Nose is bluish red in colour with dilated capillaries (**Fig. 3.101**).

Skin Adnexal Tumours

They are tumours arising from accessory skin structures like sebaceous glands, sweat glands and hair follicles. They can be benign or malignant. Clinical features are painless well localised swelling in the skin. Skin is adherent and often ulcerated. Malignant lesions are



Fig. 3.101: Rhinophyma—typical look.

nodular, hard, indurated and often fungation occurs with palpable significant regional lymph nodes. It often mimics epithelioma of skin (**Figs 3.102A and B and 3.103**).

Turban Tumour

It is a descriptive term wherein entire scalp looks like a turban because of multiple scalp swellings. It can be due to *multiple cylindroma*; *multiple hidradenomas*; *subcutaneous neurofibromas*; *nodular multiple basal cell carcinoma*. **Multiple cylindroma** is usually considered disease under this term. Cylindroma is a variant of eccrine spiradenoma (skin adnexal tumour). Multiple firm pinkish nodules in the scalp are the presentation in multiple cylindroma. **Hidradenoma** is a rare benign sweat gland tumour. Multiple tumours commonly look like a turban in the scalp. They are painless, disfiguring, cosmetically problematic soft, boggy, nonfluctuant, noncompressible cutaneous swellings; commonly observed in middle age group.

Dermatofibroma (Sclerosing Angioma, Subepithelial Benign Nodular Fibrosis, Dermal Histiocytoma)

It is a benign tumour containing 'mat like or cart wheel' pattern spindle cells arising from *dermal dendritic cells*. It presents as red or brownish yellow (due to lipid) or bluish black (due to haemosiderin), firm, single or multiple nodules occurring commonly in limbs.



Figs 3.102A and B: Benign skin adnexal tumour in the face and scalp.



Fig. 3.103: Malignant skin adnexal tumour.
Note the dilated veins.

Dermatofibrosarcoma Protuberans

It is a low grade slowly growing fibrosarcoma occurring in trunk (common site-50%), back, head and neck and abdominal wall. It is nodular, hard, with often multiple swellings with redness and ulcerations over the summit. Regional lymph nodes may get enlarged. Spread to lungs can occur only rarely. It should be differentiated from squamous cell carcinoma or skin adnexal tumour. Often there is melanin pigmentation over the surface (*Bedner's tumour*) (**Figs 3.104A and B**).



Figs 3.104A and B: Dermatofibrosarcoma protuberans in two different patients.

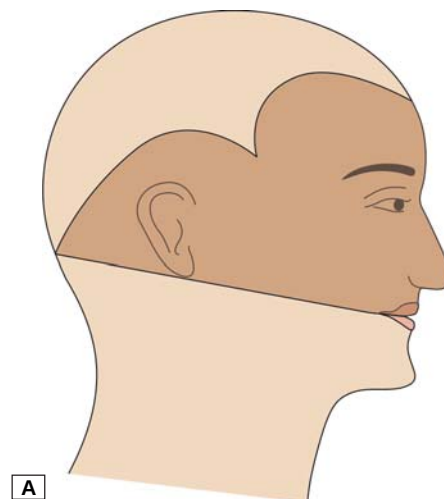
Basal Cell Carcinoma (BCC, Rodent Ulcer)

It is low grade, locally invasive, carcinoma arising from basal layer of the skin or mucocutaneous junction. It does not arise from mucosa. It is the *commonest* skin tumour. It is more common in *white skinned* people. It is common in places where exposure to ultraviolet rays is more like *Australia*. It is common in males and older people. It is common over the *face*. In the face it is common above the line drawn between

angle of the mouth and ear lobule (90%). As it is common in area where tears roll down it is called as *tear cancer*. It can occur occasionally in other parts of skin (scalp, neck, arms, and hands) or mucocutaneous junction like in anal region, genitalia. It is only *locally malignant*. It *does not* spread through blood or lymph nodes. It can *erode* deeply into adjacent deeper tissues even cartilages or bone and hence called as *rodent ulcer*. But most BCC are superficial and confined to skin. Erosion is common in lesions very close to nose or eye. It can be nodular, cystic, nodulocystic, ulcerative, multiple (associated with syndromes), pigmented, geographical/field fire/forest fire (wide area of involvement with central scabbing and peripheral active proliferating edge) or basissquamous type (combination of BCC and SCC. It behaves like SCC with regional nodal spread). BCC *never* spreads into regional lymph nodes. *Clinicopathologically* it can be superficial; morpheic or fibroepithelioma type of Pinkus. *Histologically* it contains outer columnar cells arranged in palisading manner with central polyhedral cells *without* prickle cells or keratinisation. *Clinically* it is commonly *nodulocystic/noduloulcerative* (90%), nontender, slowly growing; nonmobile if fixed to deeper plane, raised and *beaded edge* (not everted) with *central area of scabbing*. Scab repeatedly falls off and reforms. Itching over the scab can be present. Often it is disfiguring. It gives a false impression of spontaneous healing to the patient. Beads signify area of active proliferative cells. Regional nodes are *not* involved due to large sized cells. It should be differentiated from squamous cell carcinoma; melanoma; keratoacanthoma or seborrhoeic keratosis. BCC near the eye/nose/ear, BCC more than 2 cm size are called as high risk BCC (**Figs 3.105A and B to 3.107**).

Squamous Cell Carcinoma (Epithelioma, SCC)

Squamous cell carcinoma of skin arises from squamous layer (prickle cell layer) of the skin. It is the second most common skin cancer. It is common in males. It occurs usually in preexisting lesions like Bowen's disease, leukoplakia, chronic scars, chronic chemical irritation, radiodermatitis, senile keratosis, Khangri cancer in Kashmir, chimney scrotal cancer, and Kang cancer of Tibetans. It can also occur as de novo. Grossly lesion can be proliferative, ulcerative or red plaque



Figs 3.105A and B: Common site of basal cell carcinoma —above the line drawn between angle of mouth and ear lobule. Also photo showing typical location of basal cell carcinoma.

like. It is common in face, cheek, lips, hands, legs and sole. It can occur in penis, vulva, buccal cavity, tongue, oesophagus, bronchus, gallbladder, anorectum, renal pelvis and urinary bladder.

Clinical features: Ulcerative or ulceroproliferative lesion with raised and everted edge; indurated edge and base; with hard, nodular, nontender enlarged regional lymph nodes (**Fig. 3.108**). Blood spread is not common in SCC. *Marjolin's ulcer* is a well



Fig. 3.106: Photo showing features of basal cell carcinoma in the nape of neck. It is not a typical location of BCC.



Fig. 3.107: Patient with Xeroderma pigmentosa having BCC nose. These patients are also prone for cutaneous melanoma. Xeroderma pigmentosa is an autosomal recessive (chromosome 9q) disease with skin erythema, intolerance to UV rays, pigmentation and photophobia. There is defect in DNA nucleotide repair mechanism. 60% die at or before 20 years of age. Condition was first described by Kaposi in 1874.

differentiated SCC occurring in *unstable chronic scar* of long duration. It is common in scars of snake bite, venous ulcer and burns. It is only locally malignant without nodal spread. *Verrucous carcinoma* is a variant of well differentiated SCC occurring in mucous membrane or mucocutaneous junction presenting as dry, exophytic, warty, indurated growth without any



Fig. 3.108: Squamous cell carcinoma over eye destructing eyelids. Note the everted edge.

nodal spread carrying good prognosis (**Fig. 3.109**). Verrucous carcinoma of foot is called as *carcinoma cuniculatum*. Histologically malignant squamous cells with epithelial/keratin pearls are typical. More than 75% keratin pearls are well differentiated; 50-75% is moderately differentiated; 25-50% poorly differentiated; < 25% is undifferentiated.

Differential diagnoses: BCC; melanoma; keratoacanthoma; skin adnexal tumour. A rare variety of multiple self healing SCC is observed usually in face as familial autosomal dominant (chromosome 9q) disease in western Scotland—*Ferguson-Smith syndrome*.



Fig. 3.109: Proliferative cauliflower-like lesion in the foot—typical of squamous cell carcinoma.

Naevi (Mole)

It is hamartomata of melanocytes due to excessive stimulation. Few moles will be present during birth. Number increases by age. Adult has got average of 60-100 moles in the body (in Caucasians). During childhood and adolescence existing moles may get more pigmented or may regress completely. Moles are more common among Caucasians, Australians. Moles are not seen in Albinos. Moles turning into malignancy are less common in children and in Negroes. Moles are more common in limbs, face and mucocutaneous junction like mouth and anus. Mole is usually light brown or black in colour which does not fade by pressure. Moles are usually soft in consistency. Usual size of a mole is 1-3 mm. Microscopic appearance does not reflect the macroscopic/clinical look (**Fig. 3.110**).



Fig. 3.110: Congenital naevus.

Types:

Hairy mole: It is most common type which is flat or with slight raise with growing hairs on the surface. It also contains sebaceous glands and so can get infected to form a swelling which is difficult to differentiate from malignant transformation (**Fig. 3.112**).

Non-hairy mole: It is also called as smooth mole as it is not elevated but smooth, brown, pigmented lesion without hairs on the surface.

Blue naevus: It is uncommon smooth mole. It is seen in children. It is located deep in the dermis (deep intradermal) and so even though pigment is brown in colour, due to overlying thick epidermis and part of dermis fades it into blue colour. It is common in buttocks (Mongolian spot), hands and feet.

Intradermal naevus: This is the mole which consists of clusters of melanocytes in the dermis. It can be flat/raised/hairy/nonhairy. It is common in arms, face and trunk. It hardly becomes malignant.

Junctional naevus: It is centered in the junctional layer/basal layer of the epidermis as clusters of proliferating melanocytes. This type commonly turns into malignancy. It is common in palms, digits, soles and external genitalia (**Fig. 3.111**).



Fig. 3.111: Junctional naevus.



Fig. 3.112: Hairy naevus.

Compound naevus: It is combination of intradermal and junctional types. Intradermal part is inactive but junctional part is potentially malignant.

Juvenile melanoma (Spitz naevus): It is a junctional mole appearing before puberty. It is a misnomer. It is seen in face.

Hutchinson's freckle: It is seen in elderly with large area of dark pigmentation. It is common in face, neck and trunk. In the macular stage it is smooth and brown; in the tumour stage it is dark and irregular. It can turn into melanoma commonly. Malignant change is often difficult to identify clinically. It is peculiar due to its late age of onset and high chances of malignant transformation.

Halo naevus: It is depigmentation halo around the pigmented naevus. Malignant transformation in such naevus needs to be ruled out.

Spindle cell naevus: It is dense, black pigmented lesion containing spindle cells and atypical melanocytes at the junction. It is common in females with high malignant potential.

Naevus spilus (speckled lentiginous naevus): It is hyperpigmented speckles throughout. It has got low malignant potential.

Naevus of Ota: It is a dermal melanocytic hamartoma seen in the distribution of trigeminal nerve; commonly ophthalmic/maxillary divisions. It is seen in Oriental and African race adolescent females with a hormone influence.

Naevus of Ito: It is similar lesion occurring in shoulder region.

One should always remember that *it is wrong to assume all intradermal naevus remain benign and all junctional naevus will turn into malignancy*. Naevus more than 1% of body surface area or more than 20 cm in size is called as *giant naevus*. Eventhough mole and freckle are used synonymously, but by definition **mole** is increased number of melanocytes and **freckle (ephelis)** increased melanin pigment with normal number of melanocytes.

Melanoma

It is a malignant tumour arising from melanocyte which is the most aggressive cutaneous malignant tumour. It is of *neural crest* (ectodermal) origin. It is 20 times more commonly seen in *whites* than blacks. Incidence is equal in both sexes. Incidence increases over years. It is not known to occur before puberty. In females leg is the commonest site (**Fig. 3.113**). In males trunk is the commonest site. In Bantu tribe sole is the commonest



Fig. 3.113: Melanoma on the plantar aspect of the foot. Patient often will not observe this lesion. Note the deep pigmentation and ulceration over it.

site. It can occur in eyes, mucocutaneous junction, mucosa, head and neck. It is common in *Australia*. It is common in white skinned people. Exposure to ultraviolet light predisposes to melanoma. Risk factors—high society people; albinism; xeroderma pigmentosa; junctional naevus; familial dysplastic naevus syndrome; congenital naevi; family history of melanoma; previously other skin cancer if occurred.

Clinical types

Superficial spreading (64%): It is the commonest type. It has got more radial growth than vertical. It arises from preexisting naevus. It carries better prognosis. It has irregular variegated look.

Nodular melanoma (20%): It shows more vertical growth with invasion; more aggressive; common in mucosa and mucocutaneous junction; it appears as *de novo* in skin. Nodal spread is common; it is uniform and nodular; carries poor prognosis.

Lentigo maligna melanoma (10%): Less common; least malignant; common in elderly females; common in face, neck, hands. It is slow growing, *in situ* type.

Acral lentiginous melanoma (5%): Least common; common in palms and soles; common in Japan, Africa and Asia; nodular with vertical growth; attains large size; has poor prognosis; less common in whites; mimics fungal infection or pyogenic granuloma.

Amelanotic melanoma: It is worst type. Due to undifferentiation tumour cells will not synthesize melanin; rapidly progressive pinkish fleshy growth is the presentation; mimics soft tissue sarcoma.

Desmoplastic melanoma: It has got high affinity for perineural invasion; common in head and neck; carries high recurrence rate.

Subungual melanoma: It is involvement of nail fold matrix; progressive widening pigmentation of nail fold with nail dystrophy is typical—*Hutchinson's sign*. It was earlier thought of Acral lentiginous type but now considered as superficial spreading type.

Classifications

Breslow's grading: Depends on the depth/thickness of skin involvement: I—0.75 mm; II—0.76–1.5 mm; III—1.51–4 mm; IV—> 4 mm.

Clarks levels: Depends on extension into deeper plane: 1: epidermis only; 2: extension into papillary dermis; 3: entire papillary dermis is filled; 4: extension to reticular dermis; 5: extension to subcutaneous tissue.

Melanoma can be cutaneous; extracutaneous (ocular is common); occult (primary not known).

Clinical features: It can occur in a preexisting naevus or de novo in normal skin. Pigmentation with irregular surface, irregular margin; ulceration; bleeding; itching; colour changes; depigmentation halo around the pigmented area; recent increase in size.

ABCDE of melanoma: **A**symmetry; **B**order irregularity; **C**olour variation; **D**iameter > 6 mm; **E**levation. Induration is *not seen* in melanoma. Melanoma spreads through lymphatics to *regional nodes* by permeation or embolisation; through *blood* to liver (massive pigmented liver); lungs (cough, haemoptysis, pleural effusion, cannon ball secondaries); brain (convulsions, localising features, raised intracranial pressure); bones (bone pain, pathological fracture, neurological deficits); skin; viscera (melanuria). Secondary skin nodules within 2 cm of primary are called as **satellite nodules**; nodules beyond 2 cm from primary up to the regional nodes are called as '*in-transit*' nodules (Fig. 3.114). Melanoma in choroids carries better



Fig. 3.114: Melanoma in sole with satellite nodules. Satellite nodules occur within 2 cm of the primary lesion.

prognosis as there are *no* lymphatics. Late massive liver secondaries even after 20 years is known to occur when primary is specifically in choroid. Presentation initially as secondaries is possible when occult primary exists in anus, scalp, genitalia, eye, nailbed, external auditory canal, adrenal medulla.

Differential diagnoses are other pigmented lesions of the skin (**Figs 3.115A and B to 3.118**).

Pigmented lesions of the skin

- Seborrhoeic keratosis
- Dermatofibroma
- Pigmented BCC
- Naevus
- Cutaneous haemangioma
- Melanoma
- Skin adnexal tumours
- Solar keratosis
- Pyogenic granuloma
- Angiosarcoma of skin
- Café au lait patch
- Campbell de Morgan spot
- Venous dermatitis



Figs 3.115A and B: Melanoma face with extensive destruction.



Fig. 3.116: Melanoma thigh with secondaries in inguinal lymph nodes.



Figs 3.117A and B: Melanoma great toe. Note the pigmentation near the base of the toe and extensive involvement of the toe.

Sarcomas

Sarcomas arises from soft tissues (connective tissues, mesenchymal) and bone. They are *less common* than carcinomas but more aggressive. They are rapidly



Fig. 3.118: 'In-transit' nodules in melanoma. They are secondary depositions in dermal lymphatics.

growing nonencapsulated *fleshy* malignant tumours. It is 1% of adult malignancy. *Commonest site* is lower limb (35%). Commonest soft tissue sarcoma is *liposarcoma*. Usually soft tissue sarcoma attains large size more than 10 cm in 40% of cases. **Spread** is commonly to lungs through blood. *Lymphatic spread* occurs *only* in few soft tissue sarcomas like rhabdomyosarcoma, synovial sarcoma, epithelioid sarcoma, angiosarcoma.

Clinical features: Present as painless, smooth, hard, vascular, progressive swelling of short duration which causes compression and infiltration of adjacent structures. Eventually it becomes painful and tender due to nerve infiltration, infection, tumour necrosis. Features of lung secondaries like cough, chest pain and haemoptysis can be the presentations. Sarcoma occurs in *younger age group* compared to carcinomas. Skin over the swelling is stretched, glossy, with dilated veins. Very vascular sarcomas can be pulsatile. *Incision biopsy*, X-ray, CT scans of part and chest, MRI are the needed investigations. **GTNM** (Grade, Tumour size, Nodal status, Metastasis) **staging** is used.

Sarcomas are graded as *low* (liposarcoma, dermatofibrosarcoma), *high* (synovial sarcoma, rhabdomyosarcoma, angiosarcoma) and *undetermined* (leiomyosarcoma). Soft tissue sarcoma may be **superficial** means outside the superficial fascia; **deep** means deep to superficial fascia; may be within one **compartment** or involving many compartments.

Liposarcoma is the commonest soft tissue sarcoma (20% of all soft tissue sarcomas). It can occur de novo or in a preexisting lipoma. Thigh, back and retroperitoneum are the commonest sites. It can be well differentiated; myxoid; round cell; pleomorphic types.

Malignant fibrous histiocytoma (MFH) is soft tissue sarcoma with fibrohistiocytic appearance. It is common extremity sarcoma. It is seen in adults and elderly.

Leiomyosarcoma arises from smooth muscles with whorled appearance. It is common in retroperitoneum and viscera. It can occur in piloerector muscle of skin.

Rhabdomyosarcoma arises from skeletal muscle. It is common in head, neck, thigh, and arm. It is the *commonest* sarcoma in *children*. It can be pleomorphic, embryonal (seen viscera like urinary bladder), botryoidal or alveolar. It is very aggressive tumour; it can spread through lymph nodes.

Synovial sarcoma originates from synovial cells of tendon sheath, joint capsule. It occurs in younger age group; common in shoulder, thigh, and leg. It spreads through blood. In 20% cases it can spread to regional lymph nodes. It is high grade aggressive sarcoma. In 10% of patients it shows calcification.

Fibrosarcoma is next common soft tissue sarcoma after liposarcoma and malignant fibrous histiocytoma. It arises from fibroblasts.

Kaposi's sarcoma arises from vascular smooth muscles or pericytes. It is common in skin, mucous membrane, lymph nodes, or viscera. It is linked to Human Herpes virus (HHV8). It presents as *multiple reddish blue nodules* in the skin with ulceration over the nodule with lymph nodal spread. *European Kaposi's sarcoma* is common in extremity; rare in viscera; common in old age; *African Kaposi's sarcoma* is common in children and young; involves skin and lymph nodes. *Transplant associated Kaposi's sarcoma* mainly involves skin. *AIDS associated Kaposi's sarcoma* shows wide disseminated involvement with spread; it is very aggressive (**Figs 3.119 to 3.123**).



Fig. 3.119: Kaposi's sarcoma—reddish blue nodules are typical.



Fig. 3.120: Aggressive vascular sarcoma of foot.



Fig. 3.121: Soft tissue tumour left thigh.

Hamartoma, Haemangioma and Vascular Malformations

Hamartomata

Hamartano means—'I miss' (Greek) or 'fault' or 'misfire' or 'error'. It is a benign lesion with aberrant differentiation producing a mass of disorganised but



Fig. 3.122: Malignant fibrous histiocytoma—a soft tissue tumour. It is the second common type after liposarcoma.



Fig. 3.123: Synovial sarcoma. It is very aggressive sarcoma arising from tendon sheaths, joint capsule.

mature specialised cells or tissue indigenous to the particular site. It is tumour like overgrowth of tissue or tissues proper to that part. It may be single lesion or multiple lesions. Haemangiomas, lymphangiomas, A-V malformations, neural malformations are the examples.

Problems with hamartomas: Pressure symptoms locally; bleeding; infection; gigantism; cosmetic problem. Hamartoma is older terminology; now word is replaced by newer terms and definitions.

Haemangioma

It is benign vascular endothelial tumour, common in girls (3:1). It is commonly seen in skin and sub-cutaneous tissue but can occur anywhere in the body like in liver, brain, lungs or other organs. It grows rapidly in first year and 70% involutes in 7 years. Early proliferative lesion is bright red, irregular; deep lesion is bluish coloured. Involution causes colour fading, softness, shrinkage leaving crepe paper like area. Commonly it is central; common in head and neck region (60%) (**Fig. 3.124**).

Classification

- Capillary
 - Salmon patch (stork bite)
 - Strawberry haemangioma
 - Port wine stain (naevus flammeus)
- Cavernous



Fig. 3.124: Haemangioma in a child involving face extensively. Haemangioma is usually compressible. On applying continuous pressure swelling partially gets reduced and on releasing swelling comes back to original size. Cystic hygroma, aneurysms are compressible. Thrombosed aneurysm is not compressible (By Prof. Ganesh Pai MCh).

Capillary Haemangioma

Salmon patch (stork bite): It presents at birth. It commonly occurs in nape of the neck (50%), face,

scalp and limbs. It usually involves wide area of skin. It is caused by an area of persistent fetal dermal circulation. With age, it goes for spontaneous regression and disappears completely (usually in one year). Hence masterly inactivity is the treatment (**Fig. 3.125**).



Fig. 3.125: Salmon patch.

Strawberry haemangioma: It may start at birth or child is normal at birth; between one to three weeks it appears as red mark which rapidly increases in size in 3 months to form strawberry/raspberry haemangioma (**Fig. 3.126**). It contains immature vaso-



Fig. 3.126: Strawberry haemangioma.

formative tissues. There will be eventually intra-vascular thrombosis, fibrosis and mast cell infiltration. It is a *true capillary* haemangioma. It is 20 times more common than port wine stain. It is common in *white girls* (girl: boy :: 3:1). It is common in head and neck region. It is clinically *compressible*, warm with bluish surface. Bleeding can occur after minor trauma and also ulceration. It involves skin, subcutaneous tissues and often muscles also. After one year of age, it slowly begins to disappear, and *completely* in 7-8 years (70% in 7 years). It is the commonest haemangioma.

- Haemangioma in periorbital region obstructs the vision in newborn with amblyopia and if persists for 7 days causes permanent visual damage. Astigmatism also can occur
- Haemangioma in nasal area in newborn may obstruct nasal airway seriously (as newborn cannot breathe through mouth—obligatory nasal breathing)
- Skin ulceration may cause haemorrhage
- Infection can occur which may lead into sepsis, necrosis or rarely septicaemia
- Systemic steroids for 3 weeks induces involution
- Usually there is no role for surgery. Surgery is done only for retained tissue after involution.

Port-wine stain (Naevus flammeus): It present at birth and persists throughout life without any change. Spontaneous regression will not occur. It presents as smooth, flat, reddish blue/intensely purple area; common in head, neck and face; often with maxillary and mandibular dermatomes of 5th cranial nerve. Eventually surface becomes nodular and keratotic. It persists throughout life. It is actually a *capillary malformation* even though considered under haemangioma. It results from defect in maturation of sympathetic innervation of skin causing localised vasodilatation of intradermal capillaries. It is often associated with Sturge-Weber syndrome, Klippel-Trenaunay-Weber syndrome and Proteus syndrome. It needs treatment—laser (pulsed dye/diode); excision and grafting; cosmetic coverage. Often expected result is not possible by treatment (**Fig. 3.127**).

Cavernous Haemangioma

It is present at birth and consists of a multiple venous channels. Its size increases gradually and may cause problems. It often contains feeding vessels which is of surgical importance. *Sites:* Head, neck, face, limbs,



Fig. 3.127: Port wine stain (Naevus flammeus)

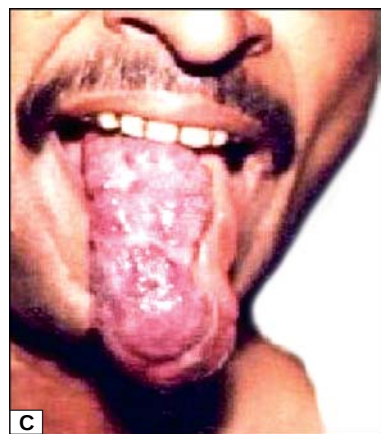
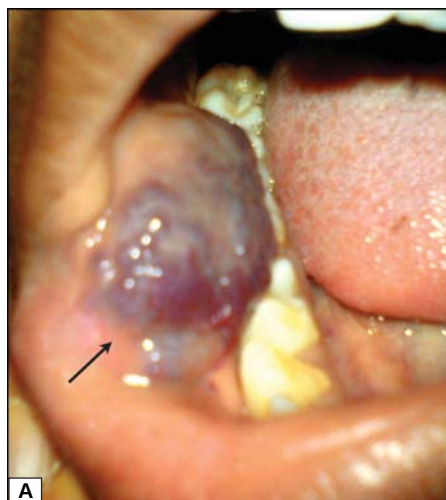
tongue, liver and other internal organs. Large or multiple cavernous haemangiomas can cause *congestive heart failure* (hyperdynamic circulation) due to shunting of large quantity of blood. Cavernous haemangioma with dyschondroplasia is called as Maffucci syndrome. Cavernous haemangioma is often mixed with lymphatic component also (mixed vascular and lymphatic).

Clinical features: It is smooth, soft, well localised, warm, fluctuant, *compressible*, nonpulsatile swelling with *bluish surface* occurring in skin and subcutaneous tissue (often in mucosa like oral cavity) without any transillumination (**Figs 3.128A to C**). *Compressibility and bluish surface* is diagnostic. When swelling is pressed it reduces partially/often completely but when pressure is released it slowly attains its original size and shape. Vascular and lymphatic malformations are compressible. It is usually nontender unless it gets infected or undergoes thrombosis or in case of haemorrhage.

Differential diagnosis: Lymphangioma—It is brilliantly transilluminant unless it is infected or fibrosed. Lipoma, cold abscess, lymph cyst—clinically it is easier to differentiate.

Complications: Haemorrhage; DIC; thrombosis; infection, ulceration and septicæmia; erosion into the adjacent bone; high output cardiac failure.

Investigations: Ultrasound; Doppler; angiogram to find out feeding vessel; platelet count; MRI/MR angiogram to see feeding vessels and deeper extension (**Fig. 3.129**).



Figs 3.128A to C: Cavernous haemangioma in the cheek near angle of the mouth; in the tip of the tongue and one involving large part of the tongue. Haemangioma tongue is one of the causes of macroglossia.

Associated syndromes

- **Klippel-Trenaunay-Weber syndrome:** Naevus flammeus + osteohypertrophy of extremities (soft tissue and bone hypertrophy) + varicose veins of lower limbs. If there is an association of arteriovenous fistula (AV fistula), it is called as **Parkes-Weber syndrome**
- **Kasabach Merritt syndrome:** Capillary haemangioma + DIC (Disseminated intravascular coagulation) with thrombocytopenia
- **Sturge-Weber syndrome:** Haemangiomas (Naevus flammeus) + hemiplegia and Jacksonian epilepsy (calcified vascular cerebral and meningeal deposits) + glaucoma.
- **Maffucci syndrome:** Cavernous haemangioma + dyschondroplasia
- **Proteus syndrome:** Naevus flammeus + regional gigantism with lymphaticovenous malformation (asymmetrical hypertrophy)
- **Osler-Rendu-Weber syndrome:** Haemangioma of skin and lip with gastrointestinal tract haemangioma (hereditary haemorrhagic telangiectasia) (autosomal dominant).



Fig. 3.129: Laparoscopic view of cavernous haemangioma of liver. It is commonest benign tumour of the liver.

Vascular Malformations

It is secondary to defect in development of vascular components, in 8th week of intrauterine period. It is associated with many syndromes. It can be in skin or in deeper planes. It is present at birth and grows in proportion to child's growth. Low flow malformations can cause skeletal hypoplasia; high flow malformations can cause hypertrophy. Consumption coagulopathy (DIC) can occur. It is equal in both sexes.

Vin Rose Patch

It is a congenital intradermal pale pink vascular malformation with dilatation of vessels in subpapillary dermal plexus. It may be associated with haemangiomas; AV malformations in limbs; congenital lymphoedema.

Parry-Romberg Disease

It is hemifacial atrophy of skin, soft tissue and bone. It is common in females. It usually begins at twenties. Atrophy of skin, fat, muscle, cartilage and bone causing *coupe de sabre* deformity—are the features. It is a self limiting disease. Aesthetic reconstruction is offered when severe deformity develops.

Campbell de Morgan Spots

It is usually smaller, elevated (2-6 mm), circular and bright red swelling. It is common in trunk; common in elderly. It is also called as *cherry angiomas*. It usually does not require any therapy.

Spider Naevus

It is an acquired solitary lesion. It contains a single dilated skin arteriole which acts like a feeding vessel; having multiple small branches in radial manner. Central arteriole of spider naevus is bright red with less prominent (less red) radiating vessels. Central arteriole is usually less than 1.0 mm in size with surrounding radiating vessels occupying around 2 mm area. It is commonly associated with alcoholic cirrhosis. Multiple spider naevi are common in face, upper arms, and proximal chest. It is completely compressible on pressure using finger or glass slide which refills entirely after releasing the pressure.

Cirroid Aneurysm

It is a rare variant of capillary haemangioma occurring in skin, beneath which abnormal artery communicates with the distended veins. It is commonly seen in *superficial temporal artery* and its branches. Here the underlying bone often gets thinned out due to pressure. It sometimes extends into the cranial cavity. Ulceration is the eventual problem which leads on to uncontrollable haemorrhage. It presents as a pulsatile swelling (*pulsating bag of worms*) in relation to superficial temporal artery, which is warm, compressible, with

arterialisation of adjacent veins and with bone thinning (due to erosion).

Lymphangioma

It is congenital localised clusters of dilated lymph sacs in the skin and subcutaneous tissue that has failed to join the normal lymph system during development period.

Simple type is also called as **capillary lymphangioma**, can be present at birth but noticeable skin vesicles often develop in few years. It is common at the junction of body to limbs—like near shoulder, axilla, groin or buttock. Skin vesicles contain clear watery or yellow fluid. Bleeding within the vesicle may turn it into brown or black. Its features includes multiple, indistinct white/brown/black coloured vesicles of 0.5 to 4 mm size at typical locations in children involving around 5-20 cm area of skin in the particular location. If it is less than 5 cm in size it is called as **lymphangioma circumscriptum** (**Fig. 3.130**). If it is more than 5 cm in size it is called as **lymphangioma diffusum**. If it is with reticulated ridges, it is called as **lymphedema ab agne**. Area is soft, spongy, often fluctuant with fluid thrill and translucency. It is not compressible. Vesicles will not fade on pressure. Often lesion may get infected to make it painful and tender. Condition will not block the lymph drainage in normal lymphatics and skin oedema is absent. Regional lymph nodes are not enlarged.



Fig. 3.130: Lymphangioma circumscripta (Courtesy Dr Balasaraswathy MD, Consultant Dermatologist, Mangalore).

Cavernous Lymphangioma

It is soft, lobulated, fluctuant, *brilliantly transilluminant* large lymphatic swelling with often multiple communicating lymphatic cysts. It often extends into deeper plane like muscle. It is common in face, mouth, lips (*macrocheilia*), tongue (*macroglossia*).

Cystic Hygroma

It is collection of clustered sequestered lymph sacs (occurring during developmental period *in utero*) presenting in newborn as large swelling which is soft, smooth, fluctuant, brilliantly transilluminant, and *compressible*. It is common in *posterior triangle of neck* (75%); axilla (20%). Rarely it can occur in cheek, tongue, retroperitoneum, groin or mediastinum. In the neck it is called as *hydrocele of the neck*. It contains *soap bubble* like aggregation of multiple cysts with larger cysts on the surface and smaller ones in the deeper plane giving a *mosaic appearance*. Cysts within are *communicating* and so make it compressible. It is lined by endothelium containing *clear* fluid which *does not* coagulate. It is present *at birth*. Due to its size it can cause obstructed labour, respiratory obstruction, rupture, infection and septicemia (**Fig. 3.131**).



Fig. 3.131: Cystic hygroma—typical site.

4

Examination of Sinus and Fistula

Sinus is a blind track lined by granulation tissue leading from an epithelial surface into the surrounding tissues. Sinus means ‘*hollow*’ or ‘*a bay*’ (Latin).

Causes: *Congenital* like preauricular sinus; *acquired* like actinomycosis, tuberculosis, pilonidal sinus, chronic osteomyelitis, median mental sinus.

Fistula is an abnormal communication between the lumen of one viscus to another or the body surface or between the vessels. Fistula means ‘*flute*’ or ‘*a pipe or tube*’. **Causes:** *Congenital* like branchial fistula, tracheo-oesophageal fistula, congenital arteriovenous fistula, umbilical fistula (patent vitellointestinal duct); *acquired* like trauma (abdomen), instrumental (during delivery), surgical, inflammatory (intestinal tuberculosis/actinomycosis), malignancy (rectovesical fistula in carcinoma of rectum). Fistula can be **external fistula** like orocutaneous; branchial fistula; thyroglossal fistula; enterocutaneous fistula; appendicular fistula or can be **internal fistula** like tracheo-oesophageal

fistula; cholecystoduodenal fistula; colovesical fistula; rectovesical fistula (**Fig. 4.1**).

Clinical Features of Sinus

Discharge from the opening of sinus—pus, caseating material, bone spicules, sulphur granules depending on the aetiology; no floor; raised often indurated edge; indurated base; nonmobile; often *sprouting granulation tissue over the sinus opening* (**Figs 4.2A and B**).

Causes of persistence of a sinus or fistula: Insufficient or nondependent drainage; foreign body or necrotic tissue underneath, e.g. suture, sequestrum, external foreign body like metal or wood pieces; persistent obstruction in the lumen, e.g. in faecal fistula, biliary fistulas (distal obstruction); lack of rest; walls become lined with epithelium or endothelium; dense fibrosis prevents contraction and healing; specific infections like tuberculosis, actinomycosis; presence of malignant disease (**Fig. 4.3**).

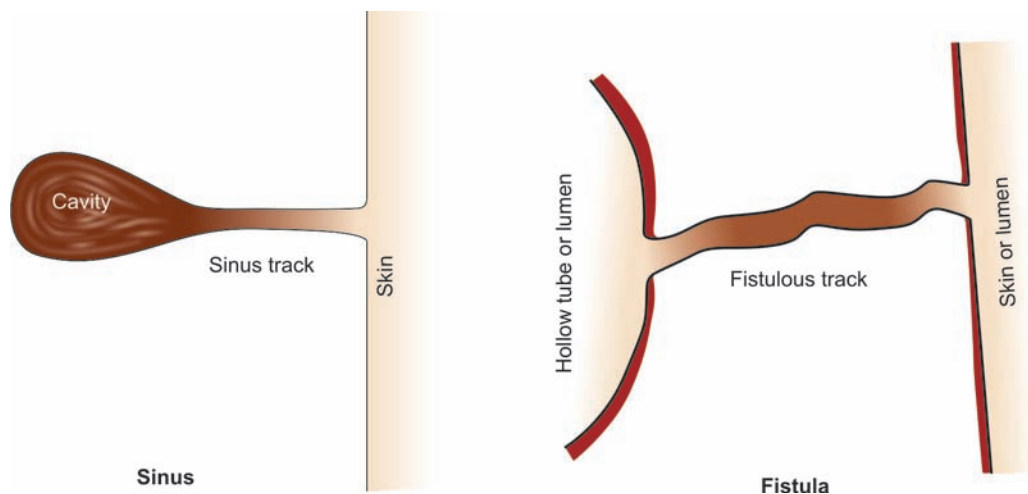
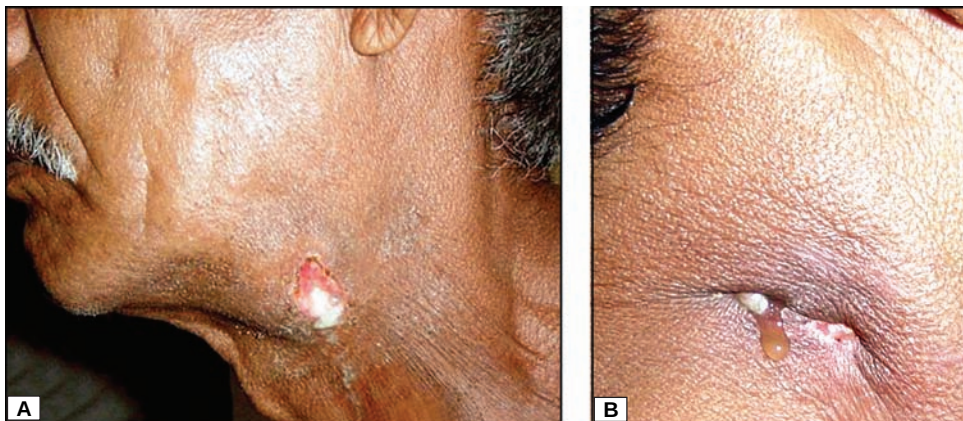


Fig. 4.1: Diagrammatic representation of sinus and fistula.



Figs 4.2A and B: Secondaries in neck causing discharging sinus (A) and (B) in two different patients.



Fig. 4.3: Fistula in ano both sides.

History

Name:

Sex:

Age: Certain sinus or fistulas are more common in certain age groups. Pilonidal sinus, branchial fistulas are common in younger age group.

Occupation:

Place:

Chief complaints: History of discharge and its duration should be mentioned. History of specific related condition also should be mentioned.

History of Present Illness

Mode of Onset and Progression

Relevant history regarding how exactly sinus has started and progressed should be asked for, whether it was healing in between and recurring again. History of trauma should be asked as osteomyelitis can occur after traumatic fracture. Detailed history about events happened prior to formation of sinus like swelling, pain, fever, deformity, difficulty in walking, etc. should be asked.

History of Discharge

Discharge is *important history* in sinus or fistula. Discharge may be purulent, yellowish/caseous like in tuberculosis, with bone spicules in chronic osteomyelitis, with necrotic material, bile/faeces/saliva/urine in different internal fistulas, sulphur granules in actinomycosis, mucus in branchial fistula, etc. Quantity of discharge, variations at different time, relation to food intake should be clarified (Discharge—quantity; quality; duration; colour; smell).

History of Pain

History of pain suggests inflammation/blockage/pus formation.

History of Fever

History of fever suggests acute/recurrent inflammation.

History Related to Associated Diseases

History related to associated diseases like of bowel disease, tuberculosis, ulcerative colitis, previous surgery, malignancy, etc. Whether patient has earlier undergone surgery like hysterectomy, with details of surgery—when it was done; immediate postoperative problem; sepsis after surgery; recovery; how long after surgery discharge or present symptom appeared. Vesicovaginal fistula may develop after hysterectomy. Faecal fistula may develop due to anastomotic leak after emergency resection and anastomosis for intestinal gangrene.

Past History

Past history of tuberculosis, Crohn's disease, actinomycosis, surgery for fistula in ano, etc. should be asked for.

Personal History

History of alcohol consumption/smoking/tobacco chewing/history of sexual contact/dietary habits are also important. *Altered appetite or weight loss* can also be mentioned under personal history—may be due to advanced malignancy or tuberculosis.

Family History

Family history of any specific diseases should be asked.

General Examination

Detailed general examination is very essential. Anaemia/oedema/jaundice/clubbing/lymphadenopathy looked for. Radial pulse/blood pressure/raise in temperature are recorded. Attitude of the patient/nutritional assessment by skin texture, subcutaneous fat, weight, body mass index/any other relevant findings should be mentioned. Increased pulse rate and temperature suggests ulcer with acute inflammation. Features suggestive of tuberculosis, spinal disease, abdominal conditions or chest disease should be looked for.

Local Examination

Inspection

Site of the Sinus or Fistula

Preauricular sinus is located in the tragus of ear or root of helix. It is directed upwards and backwards.

This occurs due to failure of fusion of ear tubercles. *Branchial fistula* occurs in the lower third of the neck. *Pilonidal sinus* occurs in sacral region. *Tuberculous sinus* is common in neck but can occur in axilla, groin, etc. (Figs 4.4 to 4.6).

Number

Usually fistulae/sinuses are single. In actinomycosis, anal fistula due to Crohn's disease and *water can perineum* they are multiple.



Fig. 4.4: Pilonidal sinus showing primary and secondary sinus.



Fig. 4.5: Discharging sinus in the neck due to tuberculosis of lymph nodes with a cold abscess underneath.



Fig. 4.6: Sinus on the scrotum could be tuberculous or syphilis or other infective focus in the testis or postsurgical cause. Tuberculosis commonly involves epididymis causing tuberculous epididymitis forming sinus on the *posterior* aspect. Syphilis involves commonly testis causing syphilitic orchitis forming ulcer/sinus on the *anterior* aspect.

Size and Appearance of External Opening

Size is small with sprouting granulation tissue. *Margin* is raised usually. In tuberculosis it is undermined thin and blue. *Sequestrum* or foreign body may extrude from the sinus.

Discharge should be inspected.

Different discharges in a sinus/ fistula: *Purulent*—bacterial infection; *creamy yellow*—staphylococcal; *watery opalescent*—streptococcal; *greenish*—*Pseudomonas*; *caseous*—tuberculous sinus; *sulphur granules*—actinomycosis; red or black granules—*Madura foot*; *mucus*—branchial fistula; *saliva*—parotid fistula; *faeces*—faecal fistula; *bile*—biliary, duodenal fistula; *bone*—osteomyelitis sinus; *anchovy sauce* like pus discharge—amebiasis cutis from amebic liver abscess. **Odour of the discharge** is also significant—smell of gas gangrene discharge is sickly—sweet odour (decayed apple); *Bacillus coli* infection in an abdominal wall sinus—objectionable odour; *Escherichia coli* discharge—odourless; *Proteus vulgaris*—proteolytic odour; bacteroides infection in abdominal wall sinus—over ripe Camembert cheese odour; faecal odour with bubbles of gas in faecal fistula

Surrounding skin should be inspected for scar/ colour/texture/dilated or visible veins/hair loss/ pigmentation/dermatitis, etc.

Palpation

Tenderness and local raise in temperature over surrounding area.

Sinus wall/margin should be palpated for induration or thickening. Chronic long-standing sinuses due to fibrosis will have thick wall.

Mobility of sinus/fistula: Most of sinus/fistula are from deeper plane; Hence are fixed and nonmobile. Occasionally superficial sinus when exists, may be mobile.

Palpate for underneath swelling which may be lymph nodes and detailed description of such swelling should be mentioned. Swelling in surrounding area should be looked for. It may be lymph nodal mass as in tuberculosis or malignant mass.

Surrounding skin, tissue and adjacent bone should be palpated for bone thickening (in chronic osteomyelitis), induration, etc. Tuberculous osteomyelitis does not show bone thickening as there is very less new bone formation (**Fig. 4.7**).



Fig. 4.7: Mandibular sinus. It is usually due to infected tooth causing osteomyelitis of mandible. It also could be due to tumour, trauma, actinomycosis and radiation. X-ray (orthopantomogram) study of discharge and biopsy are relevant investigations. Such fistula should be excised with extraction of the causative tooth.

Examination Using a Probe

Ideally probe examination of sinus or fistula should be done under general anaesthesia with all aseptic precautions and with gentleness. During probing following points to be looked for—direction, depth and length of the sinus, presence of foreign body, communication to hollow viscus in the depth (free mobility of the passed probe), fresh discharge while removing the probe.

Regional lymph node examination: In tuberculosis, infection, malignancy regional nodes may be palpable with different textures like matted in tuberculosis; hard in malignancy.

Relevant systemic examination should be done:

Examination of respiratory system is done in case of chest wall sinus. Thoracic and lumbar spine examination is done in case of psoas abscess, paraspinal abscess; urinary system examination in case of urinary fistula, loin abscess; skeletal system examination in case of osteomyelitis; digital examination of rectum in case of fistula in ano; vaginal examination in case vesico vaginal fistula; adjacent joint examination like that of hip joint in groin abscess.

Investigations

Fistulogram/sinusogram using ultrafluid lipiodol or water soluble iodine dye (lipiodol is poppy seed oil containing 40% iodine); **very essential and simple is examination of discharge** for C/S, AFB, cytology, staining (gross/physical/chemical/microscopic/staining/culture); biopsy from the edge; chest X-ray or relevant X-ray of the part like bone/joint to see osteomyelitis; ESR; CT sinusogram is very useful; MRI is most reliable in assessing the track anatomy; **three swab test** in vesico vaginal fistula (Vagina is packed with three swab, first swab high up in the anterior fornix, second one at middle of vagina, third one at lower part of vagina and 10 cc sterile methylene blue is infused into the bladder. Patient is asked to walk for 5 minutes and staining of the swab is looked for. Staining of topmost swab suggests vesicovaginal fistula or vesicocervicovaginal fistula, middle one suggests vesicovaginal fistula and lower one urethrovaginal fistula or urethral incontinence. Wetting but no staining of top most swabs suggests ureterovaginal

fistula). Contrast GI study; pyridium intake orally and looking for its excretion as coloured urine.

Classification of Sinus

Congenital—Preauricular sinus; **traumatic** with presence of foreign body; **inflammatory** like tuberculosis, osteomyelitis, chronic abscess; **neoplastic**; **other** acquired conditions like pilonidal sinus.

Commonest cause of sinus in neck is tuberculosis. Commonly it is tuberculous lymphadenitis. It shows yellowish cheesy discharge with bluish margin (**Fig. 4.8**). Usually tuberculous sinus/ulcer do not show any induration.



Fig. 4.8: Secondaries in neck with discharging sinus.

Classification of Fistula

Based on number: It may be single or multiple.

Based on type: Simple with direct track or complicated with track having variable course.

Based on opening: Lateral fistula if fistula opening is from lateral aspect of the hollow viscus; end fistula if end of the viscus opens as fistula.

Based on involvement of tissues: From viscus to skin is external; from viscus to viscus is internal.

Based on output: High output > 500 ml/day; moderate 200-500 ml/day; low output < 200 ml/day. In pancreatic fistula-high output is > 200 ml/day; low output is < 200 ml/day.

Based on aetiology: Congenital like branchial fistula; traumatic like urinary, rectal fistulas; inflammatory like appendicular fistula; neoplastic like advanced carcinoma rectum with rectovesical fistula, carcinoma cervix with uterovesical fistula, external fistula with infiltration into abdominal wall, etc.

Classification of external intestinal fistula (Irving and Beadle's): Category 1: Single orifice separate fistula with intact or healed abdominal scar may be through previous drain site (**Figs 4.9A and B**). Category 2: Single or multiple orifices close to abdominal wound or scar or any bony prominences. Category 3: Fistula (small) through a small gap of main abdominal wound. Category 4: Large fistula through a large dehiscence of bottom of main wound.



Figs 4.9A and B: Postoperative gastrointestinal fistula. Note the skin excoriation. It can be controlled by local application of zinc oxide cream.

Sequestrum

Sequestrum is dead bone *in situ*. It can be pyogenic, tubercular (feathery), salmonella (granular), syphilitic (ivory), tubular and ring (in amputation stump). It can be *unformed*—means separation between sequestrum and adjacent normal bone has not occurred or *formed*—means there is proper adequate separation between normal bone and sequestrum by forming granulation tissue. Radiologically formed sequestrum shows clear lucent area/zone of demarcation (**Figs 4.10A and B**). Sequestrum is denser because of the



Figs 4.10A and B: (A) Multiple discharging sinus with scar—osteomyelitis sinus. (B) X-ray shows sequestrum.

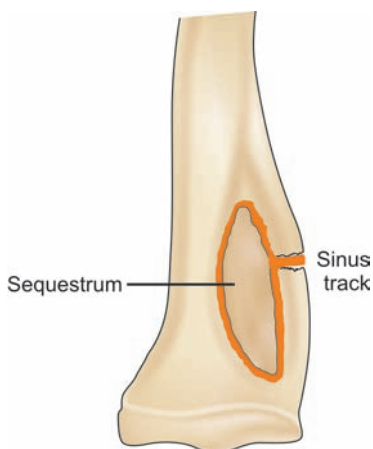


Fig. 4.11: Diagram showing osteomyelitis with sequestrum and sinus. Sequestrum is dead bone *in situ*.

absence of decalcification in the dead bone as there is no blood supply (dead bone is dense bone) (**Fig. 4.11**). Sequestrum should be formed prior to surgical intervention—sequestrectomy and saucerisation.

Median Mental Sinus

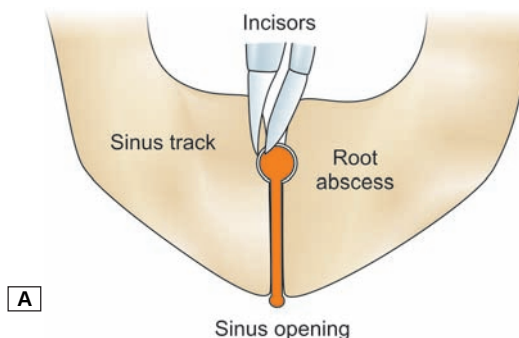
It is a chronic infective condition wherein there is infection of roots of one or both lower incisor teeth forming root abscess which eventually tracks down between two halves of lower jaw in the midline presenting as discharging sinus on the point of chin midline (**Figs 4.12A and B**).

Clinical features: It is usually painless discharging sinus in the midline on the point of chin. Often incisor infection may be revealed (in many patients clinically tooth looks normal even though root is infected invariably). It is often mistaken for infected sebaceous cyst. Osteomyelitis of the mandible is the possible complication.

Differential diagnoses are: Infected sebaceous cyst, tuberculous sinus, osteomyelitis.

Actinomycosis

It is caused by *Actinomyces israelii*. It is an anaerobic gram positive fungal like bacterium, which is a branching filamentous organism. It is called as 'ray fungus' because of sunray appearance.



Figs 4.12A and B: Median mental sinus. Note the origin of the sinus from the root/roots of the lower jaw.

Clinical types: *Faciocervical:* Infection is either from tonsil or from adjacent infected tooth. Initially an induration develops. Nodules form with involvement of skin of face and neck. It softens and bursts through the skin as **sinuses** which discharge *pus containing sulphur granules* (60%).

Thorax: Lungs and pleura get infected by direct spread from pharynx or by aspiration. Empyema develops. Later nodules appear in chest wall leading to sinuses with discharge (20%). *In right iliac fossa* it presents as a *mass abdomen* with discharging sinus. *Liver* is infected through portal vein.

Pathogenesis: Organism enters through deeper plane of the tissue, causes subacute inflammation with induration and nodule formation. Discharging sinus eventually forms over the surface. Pus collected in

a swab or sterile tube will show *sulphur granules*. Lymph nodes *are not* involved. It may cause pyaemia and may endanger life.

Investigations: Pus under microscopy shows branching filaments. *Gram staining* shows *gram positive mycelia in centre with gram negative radiating peripheral filaments*. These clubs are due to host reaction which is lipoid material.

Differential diagnosis: Chronic pyogenic osteomyelitis; carcinomas at the site; tuberculous disease.

Madura Foot (Mycetoma Pedis)

It is a chronic granulomatous condition of the foot causing multiple discharging sinuses in the foot. It was first identified in *Madurai by Gill*. It is common in India and Africa. It is common in Tamilnadu.

Organisms: *Nocardia madurae* (commonest); *Nocardia brasiliensis*; *Nocardia asteroides*; *Actinomyces israelii*.

Pathogenesis: Organism enters through a prick in the foot usually who walks barefoot → reaches deeper plane in the foot → evokes chronic granulomatous inflammation → causes pale, painless, firm nodule → vesicles form → burst to form discharging sinuses.

Features: *Discharging granules* may be *black, red, and yellow*. In *black type* of Madura foot, infection is mainly subcutaneous. In *red and yellow types*, it burrows into the deeper plane including bone causing bone necrosis (osteomyelitis). Eventually gross swelling of the limb with **multiple discharging sinuses** with disability will occur (**Fig. 4.13**). Muscles, bones, tendons and nerves are involved. Regional lymph nodes



Fig. 4.13: Madura foot. Note the multiple discharging sinuses.

are not involved. Condition will deteriorate by secondary bacterial infection.

Clinical features: Painless diffuse swelling in the foot of long duration; Later multiple discharging sinuses develop on the skin; Lymph node involvement will not occur unless secondary bacterial infection is present; Significant limb disability is common.

Differential diagnosis: Chronic osteomyelitis; tuberculous osteomyelitis; carcinoma. If infection occurs in the hand it is called as **Madura hand**. Discharge study, gram's stain, X-ray foot and biopsy are the relevant investigations.

5

Examination in Arterial Diseases

Arterial diseases commonly occur in lower limb and also occasionally in upper limb. Often both lower and upper limbs may get involved. It is often classified as lower limb ischaemia and upper limb ischaemia. But wherever is the disease detailed examination of both lower limb and upper limb vessels are required in all patients.

Name:

Age:

Sex:

Occupation:

Address:

Atherosclerosis usually occurs in old age. Thromboangiitis obliterans (Buerger's disease, TAO) occurs in young males. Even though congenital, cervical rib syndrome is seen in middle aged individuals. Raynaud's disease is common in young/middle aged females. TAO occurs commonly in lower limb. Upper limb is involved only if there is lower limb disease. Atherosclerosis involves lower limbs. Raynaud's disease occurs in upper limb. TAO and Raynaud's disease are commonly bilateral. Arterial embolism is unilateral causing sudden gangrene. Atherosclerosis often is unilateral to begin with; but eventually becomes bilateral. TAO is not observed/very rare in females. Atherosclerosis can occur in both sexes but more common in males. People working on vibrating tools/machines are prone to develop Raynaud's syndrome. Raynaud's disease is more common in women. Cervical rib is more common in females. Thoracic outlet syndrome is more often seen in swimmers, volleyball players, painters, carpenters.

Chief Complaints

Pain in the limb right/left/both—its duration.

Intermittent claudication—its duration.

Blackish discolouration/ulceration.

History

History of Present Illness

Pain

Site of pain, type of pain whether—severe burning/aching/deep persisting type is asked. Whether pain radiates (along the course of artery) or not; history of *intermittent claudication*—its duration, grade/how much distance patient can walk without pain/ whether pain subsides after walking is stopped or after continuous walk/whether patient is able to walk in spite of pain/whether there is any change in the claudication distance/site of claudication—foot/leg/thigh/buttock; Presence of *rest pain*—its location/severity/whether the pain gets relieved a little bit by holding the limb/foot/leg/toes (pain slightly lessens probably by transmission of temperature from holding hand into the part) or hanging the leg down or by applying the warmth (**Fig. 5.1**); history of pain, discomfort, colour changes when exposed to cold is especially significant in upper limb ischaemia. Application of warmth may worsen the arterial occlusion symptoms. Painful part is very sensitive and pain is precipitated/aggravated by any movement/touch or pressure sensation.

Limitation of walking—as the result of muscle pain is an important complaint.

Ulceration

Whether precipitated by trauma/spontaneous onset;



Fig. 5.1: Rest pain in a TAO patient. Observe the way patient is holding the foot to relieve the pain.

duration; progression; pain in the ulcer/type/duration/aggravating or relieving factors; type of discharge—serous-purulent-bloody should be asked.

Gangrene

Site of gangrene/its onset/progression/pain has to be asked (**Figs 5.2A to D**).

History of difficulty in walking/altered gait: Duration of such disability and progress; whether it interferes with his routine work; whether patient is bedridden due to severe symptoms has to be noted.

Mode of onset: In atherosclerosis/Buerger's disease, process of disease is spontaneous and gradual. Gangrene due to embolism is sudden in onset, rapidly progressive with radiating severe pain along the artery.

History of fever: Diabetic gangrene or wet gangrene may be associated with fever due to associated bacteraemia or localised suppuration.



Figs 5.2A to D: Gangrene toes, pregangrenous changes in some toes, gangrene leg, ischaemic changes. Always inspect the plantar aspect of the foot in all patients with peripheral vascular disease.

History of impotence: Its duration has to be asked [due to bilateral internal iliac artery (aortoiliac) block (*Leriche syndrome*)—present with pain in buttock; impotence, aortoiliac block].

History of tingling/numbness/weakness in the limbs/pins and needles sensation in the skin of foot and leg-*paraesthesia* due to shunting of cutaneous blood to deeper muscles.

History of syncope/blackouts/loss of consciousness/ blurred vision/transient ischaemic attacks (due to carotid vessel block)/abdominal colic with bloody stool—*features of involvement of other arteries*.

History of chest pain/cough or cardiac related symptoms.

History of abdominal pain/bloody diarrhoea/abdominal angina or colicky pain.

History of paraesthesia over the skin due to shunting of blood from skin to muscle.

History suggestive of superficial thrombophlebitis like swelling/redness/pain along the line of superficial vein.

Past History and Treatment History

History suggestive of similar complaints in the past; history of drug intake earlier for similar conditions like vasodilators/drugs to increase the perfusion; history of surgeries like sympathectomy/omentoplasty in the past/their results or effects are to be noted (**Fig. 5.3**).



Fig. 5.3: Lumbar sympathectomy scar with ischaemic ulcer foot showing healing sign.

Personal History

History of smoking—beedi or cigarettes/duration of smoking/number of cigarettes per day/whether smoking is discontinued and since when.

Family History

Any family history suggestive of atherosclerosis or vascular diseases or diabetes mellitus should be asked.

General Examination

Pulse-rate/rhythm/character/condition of vessel wall is noted; blood pressure of both arms and if possible of both lower limbs is checked; attitude of limbs is noted. Other detailed general examination is very essential. Anaemia/pedal oedema/jaundice/clubbing/lymphadenopathy/raise in temperature/attitude of the patient/nutritional assessment by skin texture, subcutaneous fat, weight, body mass index/any other relevant findings should be mentioned.

Local Examination

Inspection

Inspect both lower limbs keeping side-by-side as comparison is needed during clinical examination. Inspect *entire length* of the limb.

Change in colour of limb is very important sign of ischaemia. Pallor should be observed by keeping both limbs adjacent. Marked, sudden severe pallor suggests acute arterial obstruction like embolism. Presence of cyanosis/purple colour/congestion/blackish discoloration and its extent should be observed. Colour proximal to gangrene area/ischaemic area (usually ischaemic area is paler) should be noted.

Limb deformity—Its severity, gait, and attitude is noted.

Gangrene of toe/toes/foot/leg—Its extent, discharge from area, type of gangrene—dry or wet, line of demarcation—type/level/depth, colour of gangrenous area-black/purple/greenish black; reddish black [in gas gangrene (H₂S)]; odour of discharge from gangrenous area is noted.

Ulceration if any—its extent/discharge/size/shape/floor/surrounding area is noted. Patchy ulcers proximal

to gangrenous area—*skip lesions* which are usually black patchy lesions should be looked for.

Muscle wasting in the foot/leg/thigh should be observed. It should be compared with the other limb and also should be measured using a tape from a fixed bony point keeping equal distance in both limbs (**Fig. 5.4**).



Fig. 5.4: Wasting of muscles of right hand because of ischaemia. Also note the colour difference between two hands.

Features of ischaemia such as thin shiny skin/loss of subcutaneous fat/hair loss and its extent/nail changes like brittle nail/transverse ridges in the nail should be noted. Superficial small ulcerations; ulcers on pressure areas should be noted. Plantar aspect of the foot should be inspected for any infective focus/abscess/callosities/skin changes/superficial ulcers in heel/malleoli/toes (**Figs 5.5 and 5.6**).

Line of demarcation is the line between viable and non viable tissue. It is defined by a *band of hyperaemia*.



Fig. 5.5: Gangrene of all toes at their distal phalanges. All ischaemic features are obvious.



Fig. 5.6: Ischaemic changes in the right leg. 3rd and 4th toes are gangrenous with line of demarcation. Great and little toes are partly gangrenous. There are ischaemic features in the right foot and leg like hair loss/skin changes/wasting.

Line of demarcation is *well defined* in dry gangrene. It is *ill defined and unclear* in wet gangrene.

Buerger's postural test: Patient in supine position is asked to raise his legs one after another with knee kept straight. Normal limb remains pink even after 90° elevation without any pallor. Diseased limb shows marked pallor after elevation (over foot) with *empty-guttered veins*. The angle at which pallor develops (between limb and ground) is called as *Buerger's vascular angle of insufficiency*. In severe ischaemia this angle will be *less than 30°*. If foot does not become pale or when doubtful, repeated ankle flexion and extension is done until it becomes pale (*cadaveric pallor*) with empty-guttered veins on the dorsum of foot and cyanotic congestion appears after lowering the foot in 3 minutes.

On elevation pallor and blanching occurs (elevation pallor); on dependence reddish purple congestion may occur (dependency rubor).

Oedema in the foot/feet/legs suggests inflammation/congestion.

Status of the superficial veins is to be noted—normally filled veins or pale/dicoloured/guttered veins as seen in ischaemic limb.

Capillary filling time: Initially elevated limbs are made to hang down the bed. Limb will remain normal and pink in elevated as well as in dependent position because of rapid capillary filling time. In ischaemia, limb initially becomes pale on elevation and gradually becomes purple-red and then pink in more than 20 seconds. Purple-pink colour is due to deoxygenated

blood. Prolonged capillary filling time signifies severe ischaemia.

Venous refilling time: Elevated limb when laid horizontal on the bed venous refilling occurs normally within 5 seconds. It is delayed in ischaemic limb.

Palpation

Temperature of the skin is an important factor in ischaemic limb. Up to which extent the limb is cold and proximally where exactly limb/part become warmer should be assessed. Level of temperature change from distal colder to proximal warmer area is important for eventual assessment of level amputation if needed.

Tenderness— Site/extent/severity should be assessed.

Gangrenous area to be palpated for extent/whether it is dry and shriveled or whether it is wet and oedematous. Presence or absence of crepitus in gangrenous area should be checked (**Figs 5.7A and B**).

Limb above the gangrenous area should be palpated.

Capillary refilling: Tip of the nail or pulp of the finger or toe is pressed to blanch it and pressure is released (in 2 seconds) to make it pink again. Time taken for blanched area to turn pink is capillary refilling time. It is prolonged in ischaemic limb.

Harvey's venous refilling test: Two fingers are placed over the vein and pressure is applied over it. Proximal finger is moved for about 5 cm proximally without releasing the pressure. Vein between the fingers gets emptied completely and becomes flat. Distal finger is now released to see the flow of the blood and its refilling is observed, whether good or poor. It is poor in ischaemic limb.

Elevated arm stress test (EAST): Both shoulders are abducted 90 degrees with arms fully externally rotated. Patient will open and close the hands rapidly for 5 minutes. Normal individual can do this without any discomfort and pain. Patient with thoracic outlet syndrome develops pain, fatigue, paraesthesia of forearm with tingling and numbness of fingers. Patient will not be able to continue the test for 5 minutes. This test can also differentiate thoracic outlet syndrome from cervical disc prolapse disease.



Figs 5.7A and B: Ischaemic features seen in both upper and lower limbs and also gangrenes in upper and lower limb in different patients.

Roos test: Patient is asked to elevate and abduct the shoulders 90 degrees along with external rotation of arms and keep it for 5 minutes. Patient feels fatigue on the diseased side.

Costoclavicular compression manoeuvre: While palpating the radial pulse of the patient he is asked to move his shoulder backwards and downwards (exaggerated military position) which may cause absence/feeble radial pulse and a bruit may be heard while auscultating the supraclavicular region. This is due to compression of subclavian artery between clavicle and first rib.

Hyperabduction manoeuvre (Halsted test): While palpating the radial pulse, arm on the diseased side is passively hyperabducted causing feeble or absence of radial pulse. This is due to compression of artery by pectoralis minor tendon (*pectoralis minor syndrome*). An axillary bruit may be heard on auscultation.

Adson's test: While palpating the radial pulse on the affected side of the patient, patient is asked to take deep breath and turn his neck/head towards the same side so as to compress the thoracoaxillary channel. Adson's test is said to be positive when pulse becomes feeble or absent as in thoracic outlet syndrome/scalenus anticus syndrome. While taking deep breath thoracic cage moves upwards and narrows the space aggravating the compression of subclavian artery by scalenus anterior muscle. Contraction of scalenus anterior further aggravates the feature (by turning neck towards same side) (**Figs 5.8A and B**).

Branham's/Nicoladoni's sign: In arteriovenous fistula when pressure is applied over the artery proximal to the fistula, there will be reduction in pulse rate and size of the swelling with disappearance of bruit and pulse pressure becoming normal.

Allen's test: It is used in hand to find out the patency of radial and ulnar arteries. Both radial and ulnar arteries of the patient is felt and pressed firmly at the wrist. Patient clinches his hand firmly (often repeated clinching) and holds it tightly. After 1 minute clinch is released to open the palm of the hand which looks pale. Pressure on radial artery in the wrist is released to see area of distribution of the radial artery. Normally it becomes flushed with pink colour. If there is block in radial artery, the area will remain white. Test is



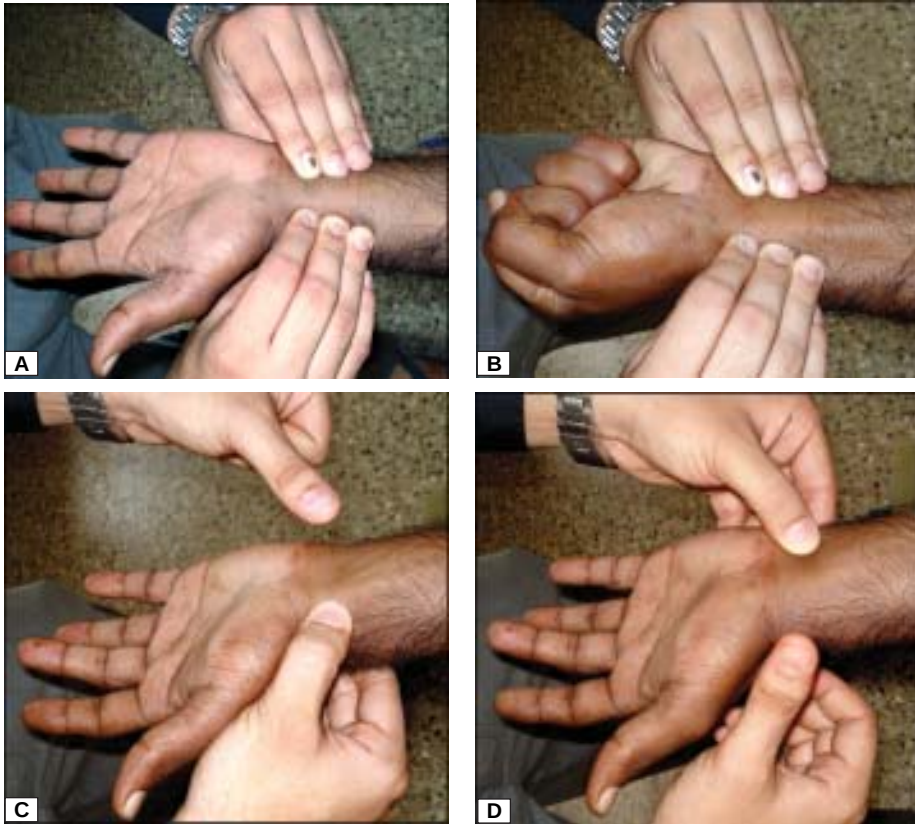
Figs 5.8A and B: Adson's test.

repeated again (**Figs 5.9A to D**). This time pressure on the ulnar artery is released to check the patency of ulnar artery. Area will be pale and blanched after releasing in case of ulnar artery block. Otherwise it becomes pink after release in normal individual.

Cold and warm water test: It is commonly done to confirm Raynaud's phenomena. Patient is asked to dip hands in cold water to precipitate the vasospasm and Raynaud's syndrome.

Crossed leg test (Fuchsig's test): Patient is asked to sit with the legs crossed one above the other so that the popliteal fossa of one leg will lie against the knee of other leg. Oscillatory movements of foot can be observed synchronous with the popliteal artery pulsation. If the popliteal artery is blocked oscillatory movements will be absent (**Figs 5.10A and B**).

Disappearing pulse syndrome: Exercise the limb after feeling the pulse. Pulse will disappear once patient develops claudication. It is because of vasodilatation and increased vascular space that occurs due to exercise



Figs 5.9A to D: Allen's test.



Figs 5.10A and B: Cross leg test—checking oscillatory movements.

wherein arterial tension can not be kept adequately and so results in disappearance of pulse (*unmasking* the arterial obstruction).

Buerger's postural test: Patient lying down on his back is asked to raise the leg forward for two minutes. In normal individuals limb (plantar aspect of foot) remains pink even after raising to 90 degrees. Ischaemic limb, when elevated shows marked pallor and empty veins. The angle in which pallor develops is called as *Buerger's angle of vascular insufficiency*. Less than 30 degrees angle indicates severe ischaemia. *Ischaemic height* of the heel in relation to the sternal angle where pallor develops in heel signifies the severity of the disease. This height in centimeter is equal to the arterial pressure in the foot in mm Hg. After that, patient is asked to keep the legs below the bed to fill the vessels. Time taken for the leg to become pink is capillary filling time. Filling time more than 30 seconds suggests severe ischaemia in the limb. In ischaemic limb, after

lowering from elevated position, cyanotic hue appears on the dorsum of foot (in 3 minutes of lowering).

Guttering of vein is observed in ischaemic limb while raising the leg for 15° due to complete collapse of the veins whereas in normal individual veins are only partially collapsed while raising the leg.

Reactive hyperaemia time test: Inflate the sphygmomanometer cuff around the limb up to 250 mm Hg for 5 minutes till significant pallor appears. Release and assess the time of appearing of red flush in skin which signifies the reactive hyperaemia time. Normal time is 2 seconds. It is delayed in ischaemia.

Palpate the limb for *crepitus* (in gas gangrene); tenderness along the line of vessel (thrombosis); oedema, etc.

Palpation of Blood Vessels

Dorsalis pedis artery is felt just lateral to the extensor hallucis longus tendon at the proximal end of first web space, against the navicular and middle cuneiform bones. It is absent in 10% cases (**Fig. 5.11**).



Fig. 5.11: Palpation of dorsalis pedis artery pulsation.

Posterior tibial artery is felt against the calcaneum just behind the medial malleolus midway between it and tendoAchilles (**Fig. 5.12**).



Fig. 5.12: Palpation of posterior tibial artery.

Anterior tibial artery is felt anteriorly in the midway between the two malleoli against the lower end of tibia just above the ankle joint lateral to extensor hallucis longus tendon (**Figs 5.13A and B**).

Popliteal artery is difficult to feel. It is palpated better (*most reliable method*) in prone position with knee flexed about 130 degrees (from straight knee 180° to 130°) to relax popliteal fascia. It is felt in the lower part of the fossa over the flat posterior surface of upper end of tibia. Artery is not felt in upper end of the fossa, as there is no bony area in intercondylar region. It can also be felt in supine position with knee flexed to 130 degrees (*most convenient method*) to relax the popliteal fossa so that pulsation can be felt over the upper part against tibial condyles (**Figs 5.14A and B**).

Femoral artery in the groin is felt just below the inguinal ligament, midway between anterior superior iliac spine and pubic symphysis (mid-inguinal point). Often hip has to be flexed for about 10-15 degree to feel it properly (**Fig. 5.15**).

Radial artery is felt at the wrist on the lateral aspect against lower end of the front of radius (**Fig. 5.16**).

Ulnar artery is felt at the wrist on the medial aspect against lower end of the front of ulna (**Fig. 5.17**).

Brachial artery is felt in front of the elbow just medial to biceps brachii tendon (**Fig. 5.18**).

Axillary artery is felt on lateral aspect of the axilla against upper end of the shaft of humerus with raised and elevated arm (**Fig. 5.19**).



Figs 5.13A and B: Palpation of anterior tibial artery.

Subclavian artery is felt against first rib just above the middle of the clavicle in supraclavicular fossa while patient is lifting the shoulder to relax deep fascia (**Fig. 5.20**).

Common carotid artery is felt medial to sternomastoid muscle at the level of thyroid cartilage against carotid tubercle (*Chassaigne tubercle*) of transverse process of 6th cervical vertebra (in carotid triangle) (**Fig. 5.21**).

Facial artery is felt against body of mandible at the insertion of masseter (**Fig. 5.22**).

Superficial temporal artery is felt just in front of the tragus of the ear against zygomatic bone (**Fig. 5.23**).



Figs 5.14A and B: Palpation of popliteal artery both in supine and prone positions. Prone position is always better.



Fig. 5.15: Palpation of femoral artery.



Fig. 5.16: Palpation of radial artery.



Fig. 5.17: Palpation of ulnar artery.



Fig. 5.18: Palpation of brachial artery.



Fig. 5.19: Palpation of axillary artery.

Condition of the vessel wall, thrill and any tenderness on the artery should be mentioned.

Ulcer if present should be examined for different features like tenderness/mobility/fixity/base induration.

Assessment of limb muscle wasting is important to find out the severity of ischaemia. It is done by inspecting the muscle bulk; prominent bony prominences; by measurement of the limb girth (circumference is measured using a tape, 15 cm away from the bony point) (**Figs 5.24A and B**).



Fig. 5.20: Palpation of subclavian artery.



Fig. 5.21: Palpation of common carotid artery.



Fig. 5.22: Palpation of facial artery.

All pulsations of both right and left side should be written in a table form

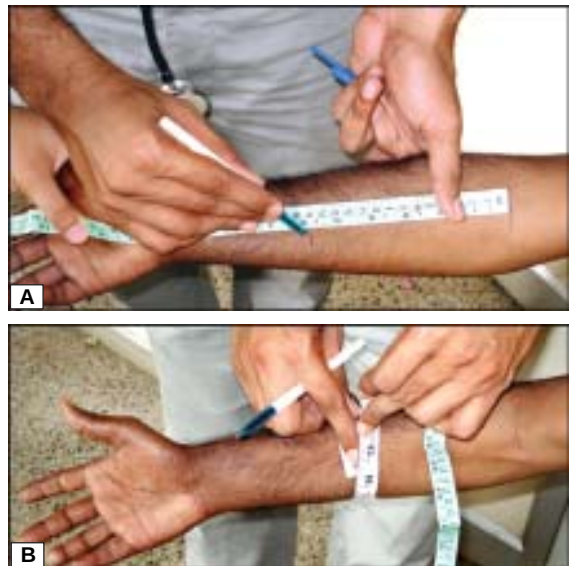
Pulse	Right	Left
Dorsalis pedis	Should be mentioned as present / absent / feeble	Should be mentioned as present / absent / feeble
Posterior tibial		
Anterior tibial		
Popliteal		
Femoral		
Radial		
Ulnar		
Brachial		
Axillary		
Subclavian		
Carotid		
Superficial temporal		



Fig. 5.23: Palpation of superficial temporal artery.

Muscle power is also checked and graded. Grade 0—complete paralysis; Grade 1—flicker of contraction, but no movement; Grade 2—movement with the elimination of gravity; Grade 3—movement against gravity, not against resistance; Grade 4—movement against partial resistance; Grade 5—normal movement against full resistance (**Fig. 5.25**).

Regional lymph node examination: In infection nodes may get enlarged.



Figs 5.24A and B: Measurement of girth is important to find out the wasting. It should be compared to opposite side and measured at a specific distance from a bony prominence.

Auscultation

Auscultation over the artery for *bruit* is done using bell of the stethoscope placed gently over the artery. It signifies localised stenosis causing *turbulent* flow. Machinery bruit/murmur is also heard in AV malformations/fistulas (**Figs 5.26A to C**).



Fig. 5.25: Muscle power should be checked against resistance to find out the grade.

Neurological Examination

When associated neurological conditions are suspected (like tabes dorsalis, syringomyelia, hemiplegia, transverse myelitis) muscle tone/power at ankle, knee and hip, sensory examination for touch, pain and temperature, reflexes at ankle and knee and plantar should be checked (**Fig. 5.27**).

Systemic Examination

Abdomen should be examined for the presence of abdominal *aortic aneurysms*. It presents as pulsatile mass above the umbilicus, vertically placed, smooth, soft, nonmobile, not moving with respiration, resonant on percussion. *Expansile* pulsation is confirmed by placing the patient in *knee-elbow* position (**Figs 5.28A and B**).

Cardiovascular system: CVS examination is essential part of the arterial system to look for any associated or causative factors. There may be embolic focus in heart like fibrillation/endocarditis, etc. (**Fig. 5.29**).

Other systems like skeletal and respiratory systems should be examined in detail.

Intermittent Claudication

Claudio means '*I limp*' a Latin word. It is a *cramp like pain in the limb muscles which is ischaemic, not*



Figs 5.26A to C: Auscultation over the major vessel like femoral/carotid for bruit is important. It signifies stenosis and turbulence flow of blood.



Fig. 5.27: Sensation should be checked for neurological deficit—especially in upper limb (cervical rib).



Figs 5.28A and B: Examination of abdomen for aortic pulsation/aneurysm; old sympathectomy scar—are important. Aortic aneurysm is looked for above the umbilicus, in midline. It shows mass with expansile pulsation; vertically placed; above the umbilicus; nonmobile; soft; smooth; resonant; retroperitoneal (does not change in position in knee-elbow position).



Fig. 5.29: Cardiovascular system examination is important to look for mitral stenosis/endocarditis, etc.

*felt prior to first step; but develops on exercise or walks and relieved by rest, develops again by similar type of exercise or walk. Due to arterial occlusion, metabolites like lactic acid and substance P accumulate in the muscle and cause pain. The site of pain depends on site of arterial occlusion. Commonest site is calf muscles. Pain in foot is due to block in lower tibial and plantar vessels. Pain in the calf is due to block in femoropopliteal site. Pain in the thigh is due to block in the superficial femoral artery. Pain in the buttock is due to block in the common iliac or aortoiliac segment, often associated with impotence and is called as *Leriche's syndrome*. Pain commonly develops when the muscles are exercising. Cause for pain is accumulation of substance 'P' and metabolites. During exercise increased perfusion and increased opening of collaterals washes away the metabolites. *Claudication distance* is distance at which claudication appears. It is very essential to assess the distance which is related to the severity of muscle ischaemia. It is better assessed using a *treadmill*. Claudication is not so common in *upper limb* but can occur in muscles of forearm and arm during writing or any upper limb exercise.*

Pain at rest; pain in tissues other than muscles; pain which does not disappear on rest—are not features of intermittent claudication.

Aortoiliac block causes claudication in buttocks, thighs, and calves; absence of femoral and distal pulses

bruit over aortoiliac region. *Impotence* occurs due to defective perfusion through internal iliac arteries and so into the penis causing erectile dysfunction (*Leriche's syndrome*). *Iliac artery obstruction* causes claudication in thigh and calf; bruit over iliac arteries with absence of femoral and distal pulses. *Femoropopliteal obstruction* causes claudication in calf with absence of distal pulses but with palpable femoral. *Distal obstruction* shows absence of ankle pulses with palpable femoral and popliteal pulses.

Boyd's classification (grading) of claudication—Three grading:

Grade I: Patient complains of pain after walking, and distance at which pain develops is called as '*claudication distance*'. If patient continues to walk metabolites causing pain are washed away in the circulation due to increased blood flow in muscle and so pain subsides by opening of the collaterals.

Grade II: Pain still persists when continued to walk; but can walk with effort.

Grade III: Patient has to take rest to relieve the pain.

Three criteria to diagnose intermittent claudication:

1. *Cramp like pain* in a muscle (e.g. calf muscle);
2. Pain develops *only* when muscle is *exercised*;
3. Pain *disappears* when exercise *stops*

Neurogenic claudication is pain in the leg during walking due to neurological causes. It often mimics vascular claudication but here arterial pulses are normal. It is common in spinal cord stenosis due to narrow canal.

Venous claudication is definitive but a rare entity; and is observed in chronic pelvic venous obstruction as a mechanical high venous pressure probably due to iliac vein thrombosis.

Rest Pain

It is continuous aching in calf or feet and toes or in the region depending on site of obstruction. It is '*cry of dying nerves*' due to ischaemia of the somatic nerves. It signifies severe decompensated ischaemia. Pain gets aggravated by elevation and is relieved in dependant position of the limb. Pain is more in the distal part like toes and feet. It gets aggravated with movements and pressure. Hyperaesthesia is commonly associated with rest pain. Rest pain is more during night time as there is reduced heart rate and blood pressure during night (sleeping time).

Fontaine classification of limb ischaemia

Stage 1: No clinical symptoms

Stage 2: Intermittent claudication

2a: Well compensated

2b: Poorly compensated

Stage 3: Rest pain

Stage 4: Gangrene, ischaemic ulcer

Critical Limb Ischaemia

It is persistently recurring ischaemic rest pain for 2 weeks or ulceration or gangrene of the foot or toes with an ankle systolic pressure < 50 mm Hg or toe systolic pressure < 30 mm Hg.

Pregangrene

It is the changes in tissue which indicates that blood supply is precarious that it will soon be inadequate to keep the tissues alive and presents with rest pain, colour changes, oedema, *hyperaesthesia* with or without ischaemic ulceration. Pallor on elevation; congestion of dependency; guttered veins; tissue tenderness; scaling of skin are the typical features (**Fig. 5.30**).



Fig. 5.30: Ischaemic ulcers in both upper and lower limbs.

Gangrene

It is *macroscopic* death of tissue *in situ* with or without putrefaction. It can occur in toes, fingers, limbs, localised area of skin and subcutaneous tissues, muscles, organs like appendix, bowel, gallbladder, testis and pancreas. It is with black/brown (colour) change; senseless/painless; pulseless (no perfusion); loss of temperature; loss of function.

Dry Gangrene

It is dry, desiccated, mummified tissue caused by gradual slowing of bloodstream. There is a line of demarcation between dead and viable tissue and is localised. It is noninfected gangrene.

Wet Gangrene

It is due to both arterial and venous block with superadded putrefaction and infection. It spreads proximally and there is no or unclear line of demarcation. It spreads faster. It is infected gangrene. It is soft and boggy.

Necrosis: It is *microscopic* cell death.

Sequestrum is dead bone *in situ*.

Slough is dead soft tissue.

Eschar is dried thick dead tissue/slough; seen in burns.

Atheroma (*Greek-gruel*) raised, focal, intimal fibrofatty plaque containing a core of lipid with fibrous cap.

Embolus (*Greek-peg*) is an abnormal, intravascular solid/liquid/gaseous material which is undissolved, transported from its site of origin to distant site/sites (**Fig. 5.31**).

Arteriosclerosis is thickening and loss of elasticity of arterial wall.



Fig. 5.31: Ischaemic bleb in the leg in a patient with peripheral vascular disease. Note patient has undergone amputation of two toes earlier.

Line of Demarcation

It is a line between viable and dead or dying tissue indicated by a band of hyperaemia. It also indicates that disease is well localised. *Final separation* or final line of demarcation between healthy and gangrenous tissue occurs by development of a layer of *granulation tissue* in between. It is hyperaesthetic due to *exposed nerve endings*.

Type of separation—Separation by *aseptic* ulceration is seen in dry gangrene. Separation by *septic* ulceration is seen in infected cases and *wet* gangrene. Rest pain, tenderness, pus discharge can occur at line of demarcation.

Note: Pressure at arterial end of capillary is 32 mm Hg; pressure at venular end of capillary is 12 mm Hg.

Features of ischaemia

Marked pallor, purple blue cyanosed appearance
Thinning of skin
Diminished hair
Loss of subcutaneous fat
Brittle nails, with transverse ridges
Ulceration in digits
Wasting of muscles
Tenderness and temperature (cold)

Features of severe ischaemia

Systolic ankle pressure less than 50 mm Hg
Systolic toe pressure is less than 30 mm Hg
Ankle brachial index is less than 0.3
Buerger's angle of insufficiency less than 20 degrees
Capillary filling time more than 30 seconds
Delayed reactive hyperaemia time
Presence of ischaemic ulcers, gangrene

Causes of ischaemic ulceration

Large artery obliteration

Atherosclerosis
Arterial embolism

Small artery obliteration

Raynaud's disease
TAO
Small artery embolism
Diabetes mellitus
Scleroderma
Vasculitis
Infective causes
Physical agents like pressure, radiation, burns, trauma

Investigations for Arterial Diseases

Blood tests: Hb%, blood sugar, lipid profile, peripheral smear, platelet count.

Doppler (*Christian Johann Doppler*—Austrian physicist) to find out the site of block.

Duplex scan: It is combination of B mode ultrasound and Doppler study. Difference in transmitted beam of the ultrasound and reflected beam is called as Doppler shift which is assessed and converted into audible signals. It is used to study the site, extent, severity of block, and also about collaterals. Audible

sound—with normal flow and sound is important. Turbulence is heard with stenosed partially blocked artery. Audible sound will be absent if there is complete block. Using Doppler probe blood pressure at various levels can be assessed. Pulse wave tracing along the artery is also important.

Plethysmography: It measures the blood flow in limbs. Water filled volume recorder; air filled volume recorder; mercury in silastic gauze is used after occluding the venous outflow. It is a noninvasive method. Segmental plethysmography using occlusion cuffs of 65 mm Hg pressure is placed at thigh, calf and ankle levels and then quantitative measure of pulsation is done.

Oscillometry: Detection of presence/poor/absence of oscillations, identify the level of block. Sudden drop in oscillations may be due to embolic obstruction. Level of amputation can be decided by this.

Ankle-brachial pressure index (ABPI): Normally it is 1. If it is less than 0.9, it means ischaemia is present. If it is 0.3 or less signifies severe ischaemia with gangrene. It may be normal at rest in early mild ischaemia but alters (reduces) during exercise. Ankle pressure is assessed by placing pressure cuff in lower calf just above the malleoli, with hand held Doppler placed over dorsalis pedis or posterior tibial artery, sound is heard during deflation of the cuff. Normal ankle pressure is very variable. A pressure less than 50 mm Hg may be critical. Toe pressure is often assessed by using appropriate sized cuffs, and by placing photoplethysmography probes on the pulp of the digit. Toe pressure less than 30 mm Hg is significant.

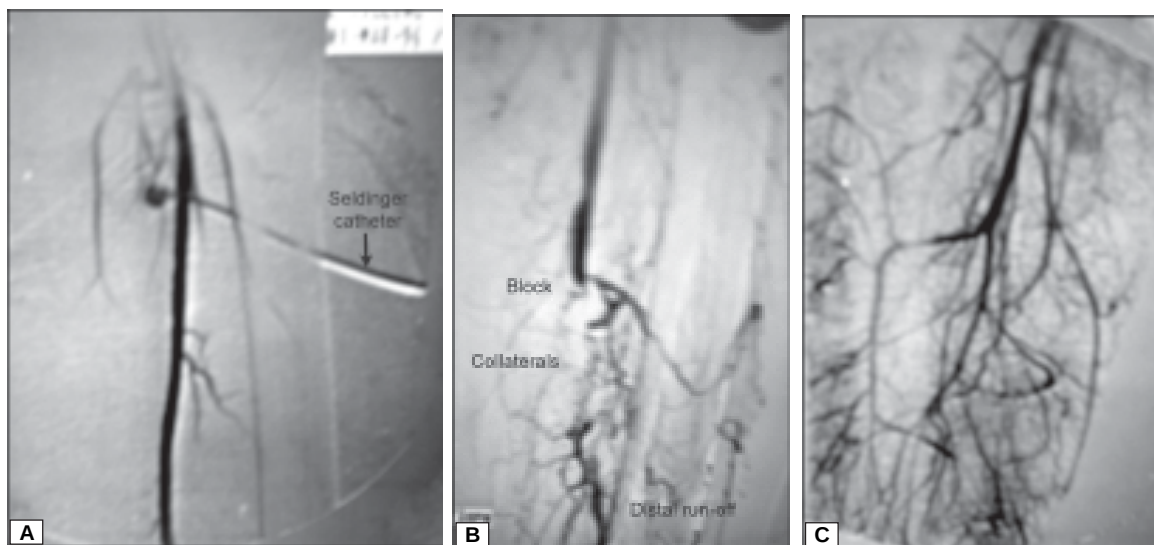
Angiography: (Enaz Moniz first did carotid angiography, 1927) Retrograde transfemoral Seldinger angiography: It is commonly done. It is done only when femorals (at least one of the femorals should be felt) are felt. If femoral pulsation is not felt then angiogram is done either *transbrachially* (left brachial artery), or *through transaortic direct puncture*. Indications for angiogram are—TAO; atherosclerosis; Raynaud's phenomenon; A-V fistulas; haemangiomas; thoracic outlet syndrome (e.g. cervical rib); aneurysms; neoplastic conditions.

Femoral artery is cannulated; needle is removed; guidewire is passed (under C arm guidance); cannula

is removed; through guidewire Seldinger (Sweden radiologist) arterial polythene catheter (5 French, 1.7 mm) is passed proximally in retrograde direction and water soluble iodine dye (Sodium diatrizoate) is injected. A trial of 5 ml is injected initially to observe iodine sensitivity. Later full dose is injected. X-rays are taken to see the block, and its extent in the affected limb. Two types of arteriography are done. Catheter tip is kept in main aorta and 30-50 ml bolus of dye is injected to see main branches and their patterns (entire arterial tree)—is called as *free flush arteriography*. If catheter tip is placed in one of the main specific artery and dye is injected—is called as *selective angiography*. In TAO *cork screw* appearance due to dilatation of vasa vasorum is characteristic. *Distal run off through collaterals* (inverted tree/spider leg collaterals); blockage—sites, extent, and severity; *severe vasospasm causing corrugated/rippled artery*—are other specific findings. *Distal run off* is amount of dye filling in the main vessel distal to the obstruction through collaterals. If distal run off is good then ischaemia is compensated. If distal run off is poor then ischaemia is decompensated. If catheter is passed still proximally angiogram of opposite side is possible. Seldinger technique can also be used (to study) to do renal angiogram to study renal artery stenosis, renal carcinomas, renal anomalies (vascular) (**Figs 5.32A to C**). *Complications* of retrograde angiogram are—bleeding; dissection of vessel wall; formation of haematoma and pseudoaneurysm; athero-embolisation into distal vessels (causes *blue toe syndrome*); thrombosis; AV fistula; infection; osmolarity discomfort (osmolarity of contrast agent is 8 times of normal plasma); vasodilatation and hypotension; nephrotoxicity; anaphylaxis (4%).

Other angiograms are carotid angiogram (direct puncture angiogram), celiac angiogram, superior mesenteric angiogram, coronary angiogram. *Direct aortic angiogram*, practiced earlier, is discouraged at present because of the risk of aortic dissection and paraplegia due to blockage of anterior spinal artery. Conventional ionic contrast agents like sodium diatrizoate is cheaper, and commonly used. Nonionic agents are costly but have less osmolarity than conventional, also have less chance of nephrotoxicity and idiosyncrasy. It is preferred in old age and diabetics.

DSA (Digital Subtraction Angiography): Here artery is delineated in a better way by eliminating other tissues



Figs 5.32A to C: *Seldinger angiogram*—retrograde femoral approach. Observe the angiogram showing block in main vessel with opened up collaterals, adequate distal run off and also showing adequate collaterals (Courtesy Dr Vasudeva Rao, Vascular Surgeon, Manipal Hospital, Bengaluru).

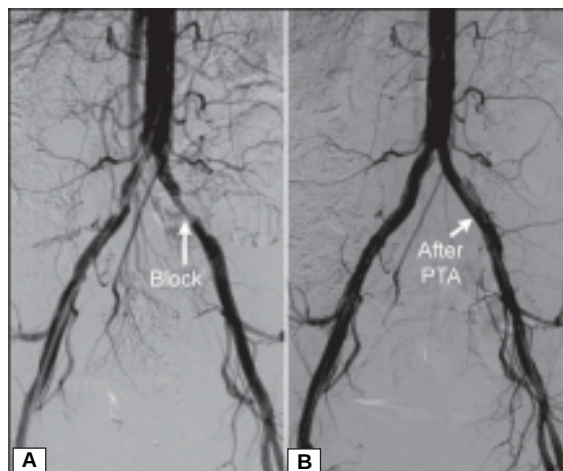
through computer system. A-V fistulas, haemangiomas, lesion in circle of Willis, vascular tumours, and other vascular anomalies are well made out. Dye is injected either to an artery or vein. Injecting into a vein is technically easier but requires larger dose of the dye. Injecting into an artery is technically difficult but small dose of dye is sufficient. **Advantages are**—Only vascular system is visualised; other systems are eliminated by computer subtraction. Small lesion, its location and details are better observed with greater clarity. **Disadvantages** are cost factor and availability. **Complications** are—anaphylaxis, bleeding, thrombosis (**Figs 5.33A and B**).

CT angiogram is very useful in aortic diseases and dissecting aneurysm.

Magnetic resonance angiogram (MRA): MRA with gadolinium enhancement [time of flight (TOF)] enhancement is very useful noninvasive method. It is the test of choice for AV malformations.

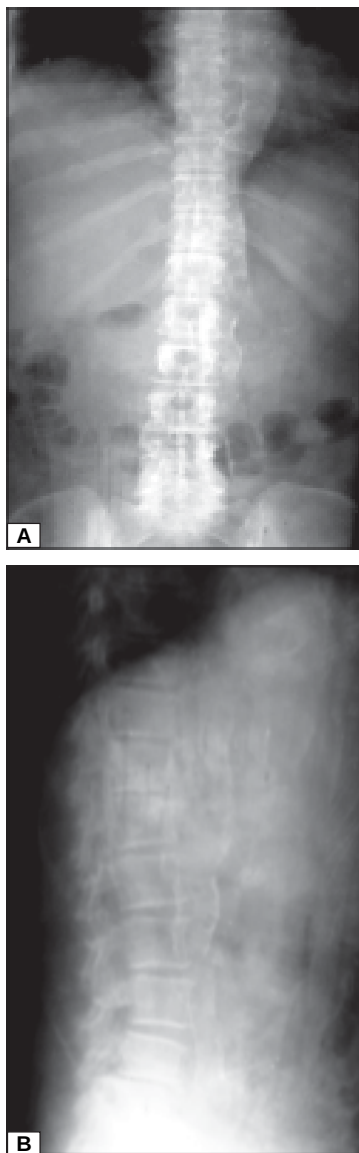
U/S abdomen: To see abdominal aneurysm or nature of aorta and other vessels.

Plain X-ray of the part: To see calcifications in atherosclerosis, Monckeberg's arterial calcification; calcification in aneurysm, cervical rib, etc. (**Figs 5.34A and B**).



Figs 5.33A and B: (A) DSA showing left sided aortoiliac block, (B) DSA after balloon angioplasty causing adequate dilatation of the left sided aortoiliac segment.

Brown's vasomotor index: Specific nerve of the ischaemic limb is anaesthetised like posterior tibial nerve or ulnar nerve (local anaesthesia or spinal anaesthesia is given to anaesthetize entire limb). If the ischaemic disease is at vasospasm stage (like in TAO), nerve block will relieve the sympathetic



Figs 5.34A and B: X-ray abdomen AP and lateral view showing calcified aorta.

vasospasm and skin temperature rises. It is compared to mouth temperature of the patient.

Rise in skin temperature—rise in mouth temperature divided by rise in mouth temperature is called as *Brown's vasomotor index*. If it is more than 3.5, it is due to vasospasm, and can be relieved by sympathectomy. If less than 3.5, sympathectomy is not beneficial.

Study of blood flow: Although specific it is less commonly used. Intramuscular injection of Xenon 133 in normal saline or Technetium 99 isotope injection is used to see the *clearance* as an assessment of blood flow in *leg muscles*. If isotope is injected intravenously, using gamma camera, direct visualisation of artery is done. Using *electromagnetic flow meter*, rate of blood flow up to 1% also can be detected. But it is technically difficult.

Transcutaneous oximetry: By placing polarographic electrodes over the skin over thigh, leg and foot oxygen tension (tcPO₂) can be measured which is reflection of underlying tissue perfusion. Normal tcPO₂ in the foot is 50–60 mm Hg. Level less than 40 mm Hg shows inadequate wound healing. Level below 10 mm Hg suggests critical ischaemia with complete failure of wound healing.

Diseases of the Arteries

Atherosclerosis

Risk factors for atherosclerosis: *Firm causes:* Hypercholesterolaemia, hypertriglyceridaemia and hyperlipidaemia; cigarette smoking; hypertension; diabetes mellitus. *Relative causes:* elderly; male; sedentary life; family history; hyperhomocystinaemia.

Atherosclerosis can cause ischaemia at various levels—foot; leg; thigh; entire limb; can be bilateral disease; upper limb ischaemia—depends on the vessel involved and extent of block it has caused (**Fig. 5.35**).

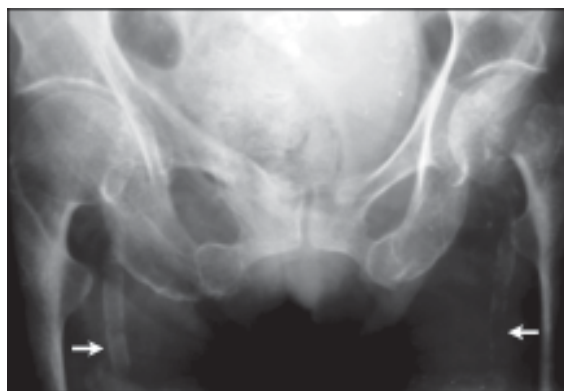


Fig. 5.35: Plain X-ray showing calcified femoral arteries due to atherosclerosis.

Thromboangiitis obliterans (TAO/Buerger's disease, Leo Buerger—Urologist)

It is a disease exclusively seen in *males* of young age group (Not seen in females due to genetic reason). It is seen only in smokers and tobacco users. Always starts in lower limb, may start on one side and later on the other. Upper limb involvement occurs only after lower limb is diseased. It is a panvasculitis.

Pathogenesis: Smoke contains *carbon monoxide and nicotinic acid* → causes initially vasospasm and hyperplasia of intima → thrombosis and so obliteration of vessels occurs. Commonly medium sized vessels are involved. *Panarteritis* is common. Usually involvement is segmental. Eventually artery, vein and nerve are together involved. Nerve involvement causes rest pain. Patient presents with features of ischaemia in the limb. Once blockage occurs, plenty of collaterals open up depending on the site of blockage, either around knee joint or around buttock. Once collaterals open up, through these collaterals, blood supply is maintained to the ischaemic area. It is called as *compensatory peripheral vascular disease*. If patient continues to smoke, disease progresses into the collaterals, blocking them eventually, leading to severe ischaemia and is called as *decompensatory peripheral vascular disease*. It is presently called as *critical limb ischaemia*. It causes rest pain, ulceration, gangrene. *Shianoya's criteria for Buerger's disease:* Tobacco use; only in males; disease starts before 45 years; distal extremity involved first without embolic or atherosclerotic features; absence of diabetes mellitus or hyperlipidaemia; with or without thrombophlebitis. Migratory superficial thrombophlebitis is common. Occasionally arteries in GI tract, heart and lungs can get involved. It is common in lower socioeconomic group. It is probably an autoimmune disease with often familial susceptibility. Claudication is common in foot and calf. Later ischaemia, rest pain, ulcers, gangrene

develop. Claudication is not common in thigh and buttock. Retrograde Seldinger angiogram shows blockage-sites, extent, severity is noted (**see Fig. 5.32**); *corkscrew* appearance of the vessel due to dilatation of vasa vasorum; inverted tree/spider leg collaterals; *severe vasospasm causing corrugated/rippled artery*; *distal run off* is amount of dye filling in the main vessel distal to the obstruction through collaterals. If distal run off is good then ischaemia is compensated. If distal run off is poor then ischaemia is decompensated (**Figs 5.36A to F and 5.37**).

Raynaud's Phenomenon

It is an episodic recurrent vasospasm, i.e. arteriolar spasm. It leads to sequence of clinical features called as Raynaud's syndrome. It is common in digits. Exposure to cold or stress causes initial pallor; later cyanosis with pain and paraesthesia; eventual hyperaemic response causes marked rubor.

Raynaud's syndrome: It is sequence of clinical features due to *arteriolar spasm*.

1. *Local syncope:* It is due to vasospasm, causing white, cold palm and digits along with tingling and numbness.
2. *Local asphyxia:* It is due to accumulation of deoxygenated blood as the result of vasospasm causing bluish discolouration of palm and digits with burning sensation (it is due to accumulated metabolites).
3. *Local recovery:* It is due to relief of spasm in the arteriole, leading to return of blood to the circulation causing flushing and pain in digits and palm (Pain is due to increased tissue tension).
4. *Local gangrene:* If spasm persists more than ischaemic time (more than one hour in upper limb), then digits go for ulceration and gangrene. Does not occur regularly but is an occasional event in the cycle.

Smoking index [SI] =	Number cigarettes smoked per day	×	Number of years of smoking
SI > 300 is a Risk factor			
Pack Years Index [PYI] =	Number of packets of cigarettes per day	×	Number of years of smoking
PYI > 40 is a Risk factor			



Figs 5.36A to F: Different types of ischaemic ulcers. Also note wasting, loss of hair, shininess and other features of ischaemia.



Fig. 5.37: Amputated 2nd toe (ray amputation) done for toe gangrene—shows good healing. It was dry gangrene.

Causes for Raynaud's phenomenon:

- Raynaud's disease:** It is seen in females, usually bilateral. It occurs in upper limb with normal peripheral pulses. It is due to arteriolar spasm in upper limb (hand) due to abnormal sensitivity to cold. Patient develops blanching, cyanosis and later flushing as Raynaud's syndrome. Occasionally if spasm persists gangrene may develop. Symptoms can be precipitated and observed by placing hands in cold water.
- Working with vibrating tools:** Like pneumatic road drills, chain saws, wood cutting, and fishermen traveling in machine boats—seen in males.
- Collagen vascular diseases:** Like Scleroderma, Rheumatoid diseases causing vasculitis (all autoimmune diseases).
- Other causes:** Cervical rib, Buerger's disease, Scalene syndrome. It is often associated with CREST syndrome (Calcinosis cutis, Raynaud's phenomenon, Esophageal defects, Sclerodactyly, Telangiectasia).

Types of Raynaud's phenomenon: *Vasospastic and Obliterative.* *Coffman criteria* for Raynaud's syndrome—episodic attacks of well demarcated reversible self limiting colour changes for 1-20 minutes (less than 320 minutes) on exposure to cold/emotional stimuli. **Raynaud's can be—Primary Raynaud's** is an idiopathic vasospastic disorder without underlying

identifiable causes. Usually there is no significant pain in primary type. Primary is probably due to increased sensitivity of alpha 2 receptors to norepinephrine; decreased nitric oxide and endothelin 1 in endothelial cells; increased serotonin and thromboxane. It is common in females and in younger age group. Usually bilateral involving all digits. **Secondary Raynaud's** is vasospasm due to some underlying causes. Significant pain will be present especially during rewarming stage. There are positive autoantibodies; equal in both sexes; occurs at any age group; need not be bilateral.

Subclavian Steal Syndrome

Following obstruction of the first part of subclavian artery, vertebral artery provides collateral circulation to the arm by reversing its blood flow. This causes *cerebral ischaemia with syncopal attacks, visual disturbances, and diminished blood pressure in the affected limb*. Symptoms will be aggravated by arm exercise (**Fig. 5.38**).

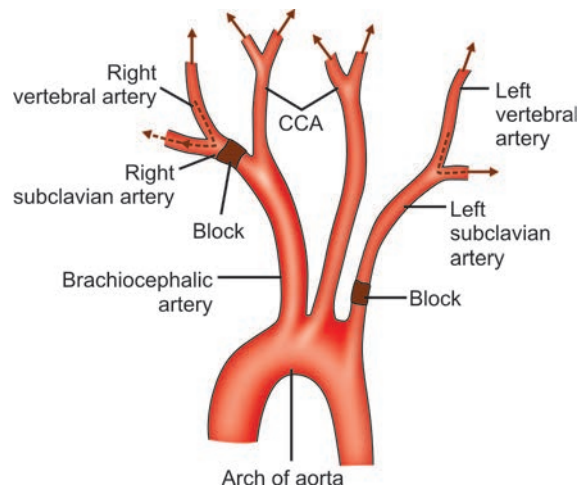


Fig. 5.38: Subclavian steal syndrome.

Takayasu's Pulseless Arteritis (Takayasu, 1938-Ophthalmologist, Japan)

It is progressive, initially symptomless panarteritis involving aortic arch and branches of aorta of unknown aetiology, probably immunological. It is common in young females (85%); common in Japan; commonly involves subclavian artery; involves all layers of

arteries of upper limb and neck; often bilateral. It remains unnoticed for long time. Fever, myalgia, arthralgia, upper limb claudication; absence of pulses in upper limb/limbs; neck; hypertension; fainting on turning the neck or change in position; atrophy of face; thrill/bruit along major arteries of upper limb and neck are the features. Optic nerve atrophy without papilloedema; weakness and paraesthesia of upper limb; cerebral softening, convulsions, hemiplegia can occur. Occasionally it can be life threatening. Myocardial infarction; embolism are other features.

Erythromelalgia/Erythralgia

It is severe burning pain and redness in the feet. Sensation of heat is so severe that patient keeps the feet in cold water to reduce it. It presents as episodic attack. There will be flushing in feet; prominent veins; warmth in the skin; severe hyperaesthesia is typical; even touching can be painful. It can be primary or secondary. *Secondary*, which is not uncommon is observed in arterial obliterative conditions, erythrocyanosis frigida, polycythaemia, gout and frostbite. *Primary* is due to unknown etiology; it is very rare.

Acute Arterial Occlusion

Causes: 1. Trauma; 2. Embolism.

Traumatic Acute Arterial Occlusion

Causes: (1) Thrombus due to trauma; (2) Subintimal haematoma; (3) Acute compartment syndrome; (4) During femoral or brachial arterial catheterisation, either for diagnostic or therapeutic procedures.

Pathophysiology: Brain tolerates ischaemia only for 4 minutes; heart for 20 minutes; limbs for 6 hours in profound acute ischaemia. Skin and bone are relatively resistant to ischaemia compared to nerves. Nervous system is most sensitive for ischaemia. When peripheral nerve is affected by ischaemia, it causes pain, paraesthesia and paralysis. Muscles play a major role in limb ischaemia as muscle accounts for the 75% of limb weight.

Clinical features: History of trauma; *pain, swelling at the site, pallor, pulselessness, cold limb (pallor).*

Acute compartment syndrome: There is sudden increase in compartment pressure more than capillary

perfusion pressure (30 mm Hg) causing impairment of tissue perfusion. It is common in anterior compartment of leg and in front of forearm. Here because of the closed compartment, pressure increases following fracture, haematoma which compresses over the vessel. It leads to blockade of vessel causing acute ischaemia of the limb presenting with severe pain, pallor, pulselessness. Measurement of *intracompartmental pressure* by placing a needle cannula directly into the compartment and using pressure transducer is ideal way to confirm the condition as Doppler still may show strong signal of pulse.

Treatment: *Immediate decompression by longitudinal fasciotomy*, is the treatment of choice, wherein deep fascia is cut adequately to relieve the compression. Otherwise limb may go for severe ischaemia, gangrene and may end with amputation. Associated fractures, haematoma, vessel tear has to be managed accordingly.

Embolism: It is due to a solid material which is floating and traveling in the bloodstream, eventually blocking the vessel on its pathway.

Arterial emboli: *Cardiac source* (80%)—due to mural thrombus following mitral stenosis and atrial fibrillation (50%); myocardial infarction (25%); others (5%). *Noncardiac* (10%)—aneurysms (5%); others (4%); paradoxical (1%). *Idiopathic* is 10%. Cervical rib causing poststenotic dilatation of subclavian artery can cause emboli. *Venous emboli* are due to DVT causing pulmonary embolism. *Fat and air embolism are other types.*

Effects of arterial embolism: Brain—Blockage at middle cerebral artery causes hemiplegia, transient ischaemic attacks (TIA), visual disturbances; Blockage at central retinal artery causes amaurosis fugax, or permanent blindness; Blockage at mesenteric vessels causes intestinal gangrene; Blockage at renal artery leads to haematuria, loin pain; Blockage at limb vessels causes pain, pallor, pulseless, paraesthesia, paresis, ulceration, gangrene. Complete sudden embolic block causes cool, waxy-white pallor whereas a partial occlusion causes pallor on elevation and rubor on dependency (**Fig. 5.39**). *Commonest site of arterial emboli is common femoral artery (40%).* Aortic bifurcation, cerebral vessel, iliac vessels account for 15% each. Upper limb and popliteal vessels are 10% each. Visceral/mesenteric is 5%.



Fig. 5.39: Fogarty catheter. It is 80 cm in length with 4 to 7 French size. It is used for embolectomy. Note the inflated balloon at the tip.

Saddle embolus: It is an embolus blocking at *bifurcation of aorta*.

Causes: Mural thrombus after myocardial infarction and mitral stenosis with atrial fibrillation; aortic aneurysm. The embolus which blocks at aortic bifurcation is usually large (**Fig. 5.40**).

Clinical features: Features of ischaemia and gangrene in both lower limbs often with neurogenic ischaemic injury.

Features of embolism

Earlier history of claudication is absent but history suggestive of disease for source of emboli will be present
Sudden, dramatic, rapid development of pain with numbness

Limb becomes rapidly cold and mottled with blebs

Loss of sensation and movements

Absence of distal pulses but forcible, expansile, prominent proximal pulse. Example – prominent femoral artery pulsation with embolic block at popliteal level

Toxic features

Fat Embolism

It is commonly seen after *fracture femur, tibia, or multiple fractures* and occasionally following electroconvulsive therapy, usually occurs in 24-72 hours. It is due to aggregation of chylomicrons, derived from bone marrow, causing fat embolism. *It is often a fatal condition.*

Features: *Cerebral:* Drowsy, restless, disoriented, constricted pupils, pyrexia, and coma. *Pulmonary:*

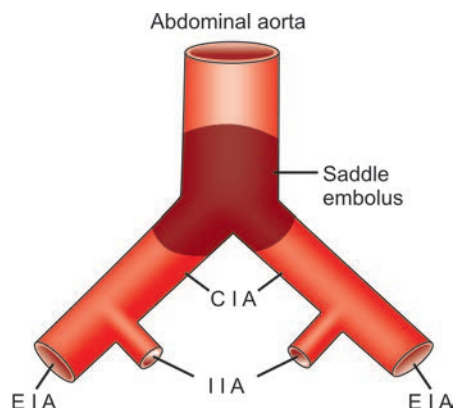


Fig. 5.40: Saddle embolus blocking the bifurcation of abdominal aorta.

Cyanosis, tachypnoea, right heart failure, froth in mouth and nostrils, fat droplets in sputum, eventually respiratory failure. *Cutaneous:* Petechial haemorrhages in the skin. *Retinal artery emboli* is the earliest sign to appear, causing *striae haemorrhages, fluffy exudates* confirmed on fundoscopic examination. *Kidney:* Blockage of renal arterioles results in fat droplets in urine.

Air embolism: **Causes:** Through venous access like IV cannula; during artificial pneumothorax; during surgeries of neck and axilla; traumatic opening of major veins sucking air inside causing embolism; during fallopian tube insufflation; during illegal abortion. Amount of air required to cause air embolism is 50 ml. When the air enters the right atrium, it gets churned up forming foam which enters the right ventricle and blocks the pulmonary artery.

Therapeutic embolisation: **Indications:** Haemangiomas; AV fistulas; Malignancies like renal cell carcinoma, hepatoma; craniovascular problems; to arrest haemorrhage from GIT, urinary and respiratory tract. In bleeding duodenal ulcer or gastric ulcer, embolisation is used to occlude gastroduodenal artery or left gastric artery respectively. It is also useful in bleeding oesophageal varices, secondaries in liver (mainly due to carcinoids), hepatoma. **Materials used are**—blood clot; human dura; gel foam; plastic microspheres; balloons; ethyl alcohol; quick setting plastics; wool; stainless steel coils.

Aneurysms

It is dilatations of localised segment of arterial system. It is due to weakening of the wall of artery. *True* aneurysm contains all three layers of artery. *False* aneurysm contains single layer of fibrous tissue as wall of the sac and it usually occurs after trauma (**Fig. 5.41**).

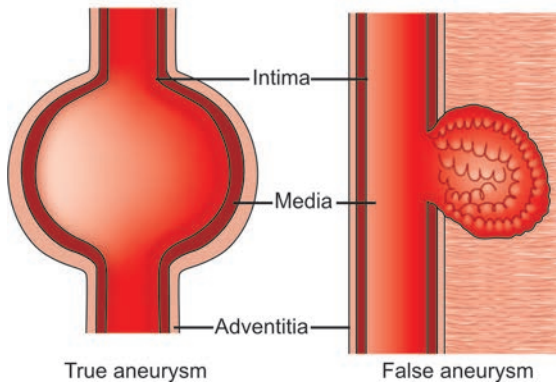


Fig. 5.41: True and false aneurysms. In true type, all layers are intact. In false type all layers breached with haematoma having a false capsule.

Types: *Fusiform*—uniform dilatation of entire circumference of arterial wall; *Saccular*—dilatation of part of circumference of the arterial wall; *Dissecting*—through a tear in the intima blood dissects between inner and outer part of tunica media of the artery (**Fig. 5.42**).

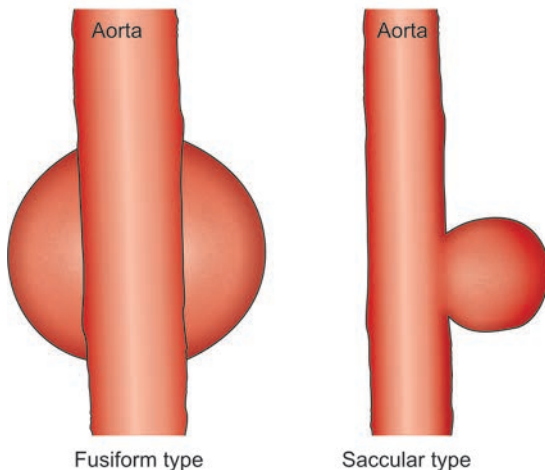


Fig. 5.42: Fusiform and saccular types of aneurysms.

Causes: Acquired: 1. *Degenerative*—Atherosclerosis (*commonest cause*); mucoid degeneration of intima and media (in South African young Negroes). 2. *Traumatic*—direct; indirect like in poststenotic dilatation by cervical rib; traumatic AV aneurysmal sac; aneurysm due to irradiation (due to dryness and destruction of vasa vasorum causing weakening of the wall). 3. *Infective*—Syphilis; mycotic; tuberculosis (in lung); arteritis; polyarteritis nodosa; acute sepsis. **Congenital:** Berry aneurysm; cirroid aneurysm; congenital AV fistula; Collagen diseases like Marfan's syndrome, *Ehlers-Danlos syndrome*.

Sites: Aorta (**Fig. 5.43**); femoral; popliteal; subclavian; cerebral, mesenteric, renal, splenic arteries. *Commonest is true, fusiform, atherosclerotic, aortic aneurysms. Berry's aneurysms are multiple aneurysms occurring in circle of Willis.*



Fig. 5.43: Thoracic aortic aneurysm.

Clinical Features of Aneurysm

1. *Asymptomatic*. 2. *Symptoms:* Swelling which is pulsatile; pain may be dull aching / severe acute type due to sudden stretching of artery / bursting type when it ruptures or forms a haematoma; referred pain due to pressing over adjacent nerves may be seen; features of ischaemia of the distal limb; painful, cyanotic distal oedema due to venous compression. 3. *On palpation:* Swelling at the site is *pulsatile (expansile)*, smooth, soft, warm, compressible, with thrill on palpation and bruit on auscultation. Swelling reduces in size when

pressed proximally; moves sideward but not along the line of artery. There is often altered sensation due to compression of nerves; erosion into bones, joints, trachea or oesophagus; aneurysm with thrombosis can throw an embolus causing gangrene of toes, digits, often extending proximally also.

Differential diagnosis: 1. Pyogenic abscess: Abscess has to be always confirmed by aspiration; especially in axilla, popliteal region, and groin. 2. Vascular tumours. 3. Pulsating tumours: Sarcomas, pulsating secondaries. 4. Pseudocyst of pancreas mimics aortic aneurysm. 5. A-V fistula.

Abdominal Aneurysms

Abdominal aortic aneurysm is the commonest aortic aneurysm. It has got 2% incidence.

Causes: Atherosclerosis: 95%. Others: Syphilis, dissecting, traumatic, collagen diseases.

Classification I: 1. Infrarenal—Commonest 95%. 2. Suprarenal 5%. **Classification II:** (1) *Asymptomatic:* Found incidentally either on clinical examination or on angiography or on ultrasound. Repair is required if diameter is more than 5.5 cm on ultrasound. (2) *Symptomatic without rupture:* Present as *back pain, abdominal pain, mass abdomen* which is smooth, soft, nonmobile, not moving with respiration, vertically placed above the umbilical level, pulsatile both in supine as well as in knee—elbow position with same intensity, resonant on percussion. GIT, urinary, venous symptoms can also occur. Hypertension, diabetes, cardiac problems should be looked for and dealt with. If aneurysm is *more than 5.5 cm then surgery is the choice*. (3) *Symptomatic ruptured aortic aneurysm:* Risk of rupture is 1%, if diameter is within 5.5 cm in size. Risk increases to 20% once the diameter ≥ 7 cm. It may be *anterior rupture (20%)* into the free peritoneal cavity causing severe shock and very early death; or *posterior rupture (80%)* with formation of *retroperitoneal haematoma* of large size causing severe back pain, hypotension, and shock, absence of femoral pulses and with palpable mass in the abdomen (**Figs 5.44 and 5.45**). Emergency management is needed.

Effects of aneurysm: Thrombosis and emboli formation; peripheral ischaemia; rupture; erosion into adja-

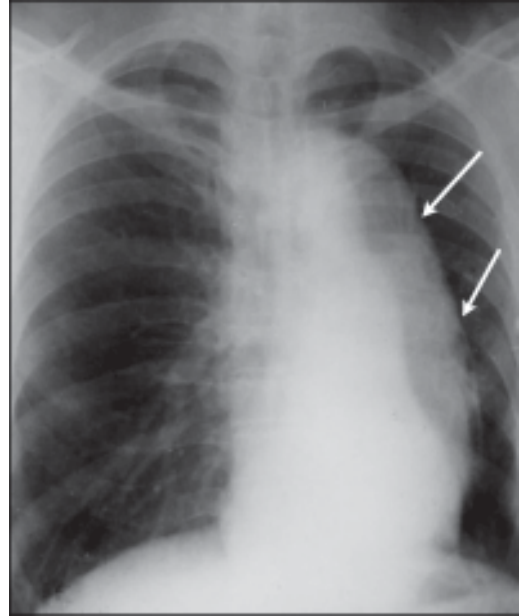


Fig. 5.44; Chest X-ray showing aortic aneurysm.

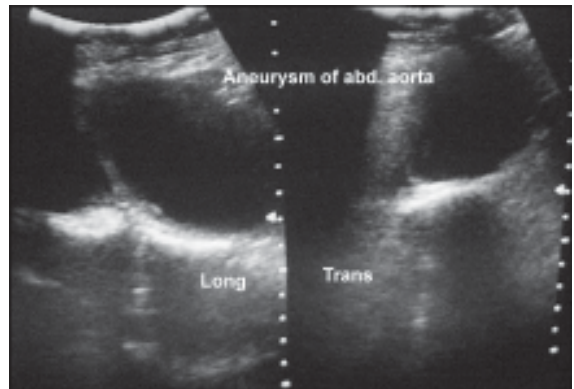


Fig. 5.45: Abdominal aortic aneurysm ultrasound picture.

cent structures like bone, bowel, pressure on organs like oesophagus (causing dysphagia); sexual dysfunction; aortoduodenal fistula; aortovenacaval fistula; spinal cord ischaemia; infection.

Peripheral Aneurysms

Popliteal aneurysm is the commonest (70%) peripheral aneurysm. It is 65% bilateral. 25% cases are associated with abdominal aortic aneurysm. 75% causes complications in 5 years. Presentations are—

swelling in popliteal region which is smooth, soft, pulsatile, well localised, warm, and compressible, often with thrill and bruit. *It may mimic a pyogenic abscess.* Thrombosis and emboli from popliteal aneurysm can cause distal gangrene which may spread proximally and may lead to amputation. Rupture may cause torrential haemorrhage.

Dissecting Aneurysm

It is the dissection of media of the aorta after splitting through intima creating a channel in the *media* of the vessel wall.

Causes: Hypertension (It is associated in 80% of dissecting aneurysms); cystic medial necrosis; Marfan's syndrome and collagen diseases; trauma; weakening of the elastic layers of the media due to shear forces. It is always seen in thoracic aorta, common in ascending aorta (70%). It is uncommon in other parts of aorta or other vessels. It can occur in aortic arch or thoracic descending aorta. This dissected aortic channel gets lined by endothelium, often re-opens distally into the aorta causing *double-barreled aorta* which in fact prevents complications. It is commonly associated with aortic insufficiency. Dissecting aneurysm is a *misnomer*. It is only *aortic dissection*. Atherosclerosis is *not a usual cause* for dissecting aneurysm.

Classification (DeBakey's): *Type I:* Dissection begins in ascending aorta extends into descending thoracic aorta (70%). *Type II:* Dissection originates and extends only up to the origin of the major vessels. It is safer type with fewer complications. *Type III:* Dissection begins in the descending thoracic aorta beyond the origin of the left subclavian artery. Dissecting aneurysm can be acute, chronic, healed dissecting aneurysm which communicates distally again to aorta as *double barreled aorta*.

Complications: *Acute:* Rupture into the pericardium or pleura—dangerous type. *Chronic:* Blockage of coronary vessels, major vessels like carotid, subclavian arteries with aortic insufficiency.

Clinical features: Pain in the chest, back which is excruciating. Features of ischaemia develop due to blockage of different vessels (**Fig. 5.46**).

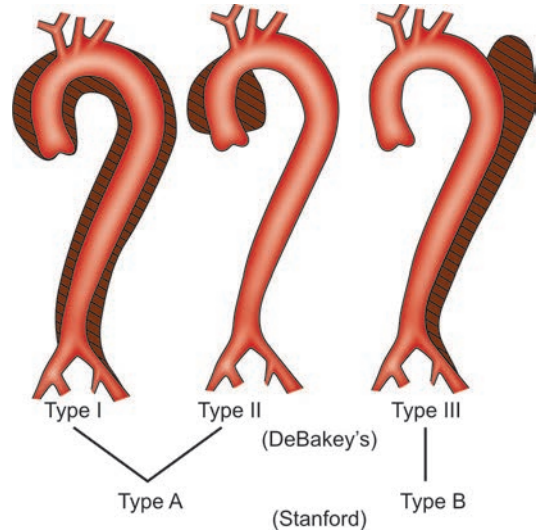


Fig. 5.46: Dissecting aneurysm.

Mycotic Aneurysm

It is a *misnomer*. It is not due to fungus but due to *bacterial* (commonly *Staphylococcus*, *Streptococcus*) infection. Origin of bacteria may be from any site of infection in the body. Common aetiology is bacterial *endocarditis* but could be any infective site. Common vessels involved are aorta, visceral, head and neck and intracranial. Commonly it is saccular, multilobed, with a narrow neck. Patient presents with fever, toxæmia and tender pulsatile mass if it is in the periphery.

Acrocyanosis (Crurum Puellarum Frigidum)

It is persistent, painless cyanosis seen in fingers and often in legs with paraesthesia and chilblains affecting young females. It is chronic persistent arteriolar constriction with slow rate of blood flow. Trophic changes, ulcerations are not seen. Cyanosis *which is persisting* may aggravate on exposure to cold. It may be associated with endocrine dysfunction. Vasodilators, and cervical sympathectomy may be effective.

Gangrene

It is *macroscopic death* of tissue *in situ* (in continuity with adjacent viable tissue) with or without putrefaction. It can occur in—limbs, appendix, bowel, testes, gallbladder.

Types of Gangrene

Dry gangrene is due to slow gradual loss of blood supply to the part causing dry, desiccated, wrinkled, mummified part with proper line of demarcation (**Fig. 5.47**). **Wet gangrene** is due to infection with putrefaction, causing oedematous, swollen, discolouration, spreading proximally, with vague line of demarcation



Fig. 5.47: Dry gangrene of leg. Patient needed above knee amputation.

Causes

1. Secondary to arterial obstruction like atherosclerosis, emboli, diabetes, TAO, Raynaud's disease, ergots etc. 2. Infective: Boil, carbuncle, gas gangrene, Fournier's gangrene, cancrum oris. 3. Traumatic: Direct/indirect. 4. Physical: Burns, scalds, frostbite, chemicals, irradiation, electrical. 5. Venous gangrene.

Clinical Features

Colour changes: Pallor, grayish, purple, brownish black discolouration due to disintegration of haemoglobin to sulphide; **Absence of pulse;** **Loss of sensation;** **Loss of function;** **Loss of temperature;** **Line of demarcation** between viable and dead tissue by a band of hyperaemia and hyperaesthesia with development of a layer of granulation tissue. **In dry gangrene** separation occurs by aseptic ulceration with minimum infection and gangrene is dry, and mummified. **In moist gangrene** separation takes place by septic ulceration. Often demarcation is vague with **skin lesions** more proximally and so ends up with higher level of amputations. Even after amputation skin flap may show **die back process**, leading to failure of taking up of amputation flap and so requires still higher level of



Figs 5.48A and B: Gangrene both in lower and upper limbs. Note all features of gangrene.

amputation. **Proximal ischaemic features** may be present with rest pain, colour changes, hyperaesthesia – pregangrene (**Figs 5.48A and B and 5.49**).

Diabetic Foot and Diabetic Gangrene

Foot is a complex structure with many layers of muscles, ligaments, joints, arches, fat, thick plantar fascia, vascular arches, neurological system which maintains weight bearing, gravity, normal walk (swing, and stance phases).

Problems in diabetic foot: Callosities; ulceration; abscess and cellulitis of foot; osteomyelitis of different bones of foot like metatarsals; cuneiforms; calcaneum;



Fig. 5.49: Below knee amputation stump which is also ischaemic. Patient underwent below knee amputation.

diabetic gangrene; arthritis of the joints. **Meggitt's classification of diabetic foot:** Grade 0: Foot symptoms like pain, only. Grade 1: Superficial ulcers. Grade 2: Deep ulcers. Grade 3: Ulcer with bone involvement. Grade 4: Forefoot gangrene. Grade 5: Full foot gangrene.

Pathogenesis of diabetic foot/ gangrene: High glucose level in tissues is a good culture media for bacteria. So infection is common. *Diabetic micro-angiopathy* causes blockade of microcirculation leading to hypoxia. *Diabetic neuropathy:* Due to sensory neuropathy, minor injuries are not noticed and so infection occurs. Due to motor neuropathy, dysfunction of muscles, arches of foot and joints, and loss of reflexes of foot occurs causing more prone for trauma, abscess, etc. Due to autonomic neuropathy, skin will be dry, causing defective skin barrier and so more prone for infection. *Diabetic atherosclerosis* itself reduces the blood supply and causes gangrene. Thrombosis can be precipitated by infection causing infective gangrene. Blockage occurs at plantar, tibial and dorsalis pedis vessels. *Increased glycosylated haemoglobin* in blood causes defective oxygen dissociation leading to more hypoxia. At tissue level there will be increased glycosylated tissue proteins, which prevents proper oxygen utilisation and so aggravates hypoxia.

Clinical features: Pain in the foot; ulceration; absence of sensation; absence of pulsations in the foot (posterior tibial and dorsalis pedis arteries); loss of joint movements; abscess formation; change in temperature and colour when gangrene sets in (**Fig. 5.50**).



Fig. 5.50: Diabetic leg severely infected with necrosis which needs amputation.

Infective Gangrene

It is development of gangrene due to infection commonly bacterial occasionally by specific organisms.

Cancrum oris; gas gangrene; carbuncle; Fournier's gangrene are the examples. It is due to infective thrombosis of local end arteries causing gangrene of the tissues and skin of that particular area.

Cancrum oris (Noma) is an infective gangrene, a severe form of Vincent's acute ulcerative gingivitis and stomatitis. It is seen in poorly nourished, ill child due to *Borrelia vincentii* and *Fusiformis fusiformis* bacteria. It starts in gums, spreads into cheek, bone, soft tissues and skin causing extensive tissue loss; destruction of mucous membrane, soft tissues, bone and skin with severe toxæmia. There will be other secondary infection also. Fetid odour, excessive salivary secretion is usual. Phagedena is destructive ulceration with gangrene seen in cancrum oris and chancroid. In phagedena there is destruction without proliferation whereas in malignancy there is destruction with proliferation. Condition has high mortality.

Gas Gangrene

It is an infective gangrene caused by clostridial organisms involving mainly skeletal muscles. Earlier it was called as *malignant oedema*. **Organisms—***Clostridium welchii* (Perfringens) a gram negative, central spore bearing, nonmotile, capsulated organism; *Clostridium oedematiens*; *Clostridium septicum*; *Clostridium histolyticum*. *Clostridium welchii* produce toxins—*Alpha* (Commonest); *Beta*; *Epsilon*; *Iota*. Various strains include—A, B, C, D, E. 'A' strain is commonest. **Exotoxins:** Lecithinase is important toxin which is haemolytic, membranolytic and necrotic causing extensive myositis. Haemolysin causes extensive haemolysis. Hyaluronidase helps in rapid spread of gas gangrene. Proteinase causes breaking down of proteins in an infected tissue. Spores enter through the devitalised tissues commonly following road traffic accidents and crush injury, etc. Spores germinate → Released bacteria will multiply → **Exotoxins** are released → cause their effects.

Effects: Extensive necrosis of muscle with production of gas H_2S which stains brown or black → usually muscle gets involved from origin to insertion → Often may extend into thoracic and abdominal muscles → When it effects the liver it causes necrosis with frothy blood → *foaming liver*, is characteristic.

Clinical features: Incubation period is 1-2 days. Features of toxæmia, fever, tachycardia, pallor are common. Wound is under tension with foul smelling discharge and *Khaki brown* coloured skin due to haemolysis. Exposed muscle is typically brick red/green/black coloured. *Crepitus* can be felt. Jaundice may be ominous sign and also oliguria signifies renal failure.

Clinical types: *Fulminant type* causes rapid progress and often death due to toxæmia, renal failure or liver failure or MODS. *Massive type* involves whole of one limb containing fully dark coloured gas filled areas. *Group type:* Infection of one group of muscles like extensors of thigh, flexors of leg. *Single muscle type* will be affecting one single muscle. *Subcutaneous type* of gas gangrene involves only subcutaneous tissue (i.e. superficial involvement). X-ray will show **gas** in muscle plane or under the skin. Once a ward or operation theatre is been used for a patient with gas gangrene, then it should be fumigated properly for 48 hours to prevent the risk of spread of infection to other patients (**Fig. 5.51**).



Fig. 5.51: Gas gangrene.

Frostbite

It is due to exposure to cold wind or high altitude (below freezing point). It is common in old age during cold spells. There is arteriolar spasm; protein denaturation; cell destruction. Damage to vessel wall causes oedema, blistering, *deep ulcer* and *gangrene* formation. Part is painless and waxy.

Treatment: *Gradual warming is done.* Part should be wrapped with cottonwool and rested. Warm drinks, analgesics, paravertebral injections to sympathetic chain, *hyperbaric oxygen* are effective.

Chilblains also called as *perniosis* is due to exposure to intense cold causing cutaneous arteriolar constriction with *superficial ulcers*.

Ainhum

A fissure develops at the interphalangeal joint of the toe which becomes a fibrous band that encircles the digit causing necrosis and gangrene of toe. It is common in males but can occur in females. History of barefoot walk during childhood is common. It is common in blacks; common in Negroes; it is often bilateral; common in 5th toe; can occur in other toes also. Dry gangrene with a constriction band / groove leading into autoamputation is common (**Figs 5.52A and B**).



Figs 5.52A and B: Typical Ainhum. Note it is bilateral. Note the constriction ring in the little toe. It may go for autoamputation. It needs Z plasty. It is common in blacks and Negroes.

Morvan's Disease

It is painless whitlow seen in fingers in syringomyelia. It is neuropathic ulceration / gangrene. Olivier d' Anger described syringomyelia in 1824 wherein there is formation of cavity in spinal cord along with fourth ventricle with thinning of neural tissue component. It causes sensory disturbances in upperlimb; weakness of hands; loss of pain and temperature sensation in hands; progressive kyphoscoliosis. Hindbrain herniation may lead into ataxia, spasticity, headache, lower cranial nerve palsy. It may be associated with Type I Arnold-Chiari malformation.

Reperfusion Injury

It occurs after reestablishment of arterial flow to an ischaemic tissue bed which further leads to tissue death. It is due to sudden release of oxygen free radicals which blocks the microcirculation, with release of high levels of potassium and myoglobin. Haemodynamically patient becomes unstable with lactic acidosis, intracellular changes, interstitial oedema and cardiac dysfunction. Severe ischaemia causes oedema in the muscular compartment with raise in compartment pressure more than the essential capillary perfusion pressure causing *acute compartment syndrome*. It is common in the anterior compartment of the leg. Acidosis, acute renal failure and cardiac arrhythmias set in and becomes life threatening. Features are – toxaemia; oliguria; persistent pain and oedema in the leg with muscular tenderness; raised blood urea and serum creatinine with features of acute ischaemia in the limb.

Treatment—Mannitol to prevent renal failure; fluid therapy; fasciotomy to reduce raised compartment pressure; antibiotics. Condition may be life threatening.

Upper Limb Ischaemia

It is a rare entity compared to lower limb ischaemia but important because of its difficulty in managing. *Higher-level amputations are rare in upper limb ischaemia.*

Causes—Thoracic outlet syndrome; Raynaud's disease and phenomenon; embolism due to causes like atrial fibrillation and endocarditis; trauma; TAO upper limb (along with lower limb TAO); atherosclerosis of upper limb vessels; Takayasu's arteritis; Polyarteritis nodosa; Scleroderma.

Features—Upper limb claudication and ischaemic rest pain; ischaemic features; ulcers and gangrene commonly in fingers; wasting of hand and forearm muscles; mass in the neck, bruit in the neck in supraclavicular region; Adson test, hyperabduction test, Roos test, Allen's tests are important.

Investigations—Arterial Doppler; subclavian angiogram; investigations to confirm vasculitis; blood sugar; lipid profile; cardiac evaluation.

Upper limb ischaemia

Trauma / cervical rib are the common causes
Opposite limb, lower limbs should be examined
Cardiovascular system should be examined
Neck should be examined
Wasting / girth should be checked
All relevant clinical methods are equally significant
Auscultation over neck / axilla / carotids for bruit are important
Doppler; angiogram; nerve conduction studies; CT neck and thorax are essential investigations
Arterial repair; therapy for cervical rib; scalenotomy; cervical sympathectomy are the different modalities of treatment (**Figs 5.53A and B and 5.54**)
Digital amputation may be required



Fig. 5.53: Cervical sympathectomy scar (done for upper limb ischaemia) in the neck/ patient has developed Horner's syndrome. Healed ulcer area over tip of index finger on left side is seen. Patient is asymptomatic after sympathectomy. Ptosis—typical look in another patient.

Arteriovenous Fistula (AVF)

It is an abnormal communication between an artery and vein. It can be **congenital** (arteriovenous malfor-



Fig. 5.54: Gangrene on left index finger. Note patient has already undergone left cervical sympathectomy.

mation) or **acquired** (usually traumatic) or **iatrogenic** (Cimino fistula done for haemodialysis).

Sites: Limbs, either part or whole of the limb may be involved. Part may be in toes or fingers; lungs; brain in circle of Willis; other organs like bowel, liver.

Clinical features: *Structural changes* in the limb: Limb is *lengthened* due to increase blood flow since developmental period; limb *girth* is increased (Robertson's giant limb); limb is *warm*; continuous *thrill* and continuous machinery *murmur* is heard all over the lesion; dilated arterialised *varicose veins* are seen due to increased blood flow and due to valvular incompetence; often there will be *bone erosion* or extension of AVF into the bone as such. *Physiological Changes:* Because of the hyperdynamic circulation, there will be *increased cardiac output* and so often *congestive cardiac failure*.

Complications: Haemorrhage; thrombosis; cardiac failure.

Acquired Arteriovenous Fistula (AVF): Causes: 1. Trauma in femoral; popliteal; brachial; wrist; aorta venacaval; abdomen - It may be following road traffic

accidents, penetrating wounds, cock-fights injury (Common in South India). 2. After vascular surgical intervention for major vessels. 3. Therapeutic: AVF is created (**cimino fistula**) for renal dialysis to achieve arterialisation of veins and also to have hyperdynamic circulation so that adequate venous access can be achieved for long time haemodialysis. Common sites are wrist, brachial and femoral region.

Pathophysiology: Physiological changes: *Cardiac failure* due to hyperdynamic circulation. *Structural changes:* Changes *at the level* of fistula—Blood flows from high pressure artery to low pressure vein causing diversion of most of the blood. Between the artery and vein, at the site of fistula, dilatation develops with fibrous sac formation called as *aneurysmal sac*. This presents as warm, pulsatile, smooth, soft, compressible swelling at the site with *continuous thrill* and *continuous machinery murmur*. It is warm at the site. *Changes below the level of the fistula*—Because of diversion of arterial blood *distal part becomes ischaemic*. Because of arterialisation of high pressure veins, and valvular incompetence, it results in *varicose veins*. *Changes proximal to the*



Figs 5.55A to E: Congenital arteriovenous malformation involving right upper limb, axillary region and part of right half of neck and face. Note the limb lengthening and widened girth. Limb is warmer. Limb should be measured at each level. Auscultation reveals continuous bruit over the limb. Note the dilated tortuous veins due to hyperdynamic circulation.

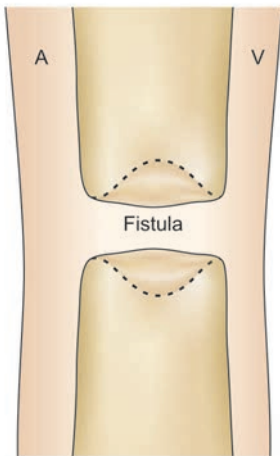


Fig. 5.56: Arteriovenous fistula diagrammatic representation.



Fig. 5.57: Acquired arteriovenous fistula in the wrist over radial vessels. It should be palpated for compressibility and thrill. It should be auscultated for bruit.



Fig. 5.58: AV fistula done for haemodialysis (Cimino fistula).

fistula—Hyperdynamic circulation causes cardiac failure. If pressure is applied to the artery proximal to the fistula, swelling will reduce in size, thrill and bruit will disappear, pulse rate and pulse pressure

becomes normal. This is called as *Nicoladoni's sign* or *Branham's sign*. Cardiac failure may be very severe in traumatic AVF (Often resistant to drug therapy) (**Figs 5.55A to E to 5.58**).

6

Examination in Venous Diseases

Writing a Case Sheet for Varicose Veins (Long Case)

Name:

Address:

Age:

Sex:

Occupation: Varicose veins are more common in people who stand for long hours like bus conductors, nurses, doctors, surgeons, manual labourers, watchmen, athletes, traffic policemen, etc. Occupation also may exacerbate the condition.

Varicose vein is more common in females (10:1). It is not commonly seen in Africa.

Chief Complaints

Pain in the leg/thigh/foot of significant duration present on one or both side.

Swelling/dilated veins in the leg of significant duration.

Pigmentation/ulceration in the leg with duration (**Fig. 6.1**).

History

History of Present Illness

Pain

History of pain in the leg/foot/or thigh with duration should be noted. Origin of pain and its severity, nature of onset whether acute or insidious should be asked. Character of pain— *dull aching* or cramping should be asked. Whether pain gets aggravated by walking/standing should be noted. Dull aching pain along the line of the vein is typical and usually gets aggravated



Fig. 6.1: Varicose vein leg of great/long saphenous vein. It is tortuous, elongated dilated vein.

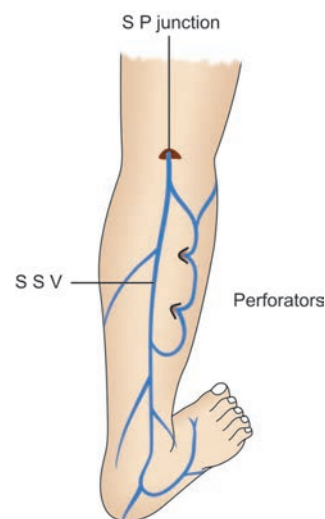
in the evening and relieved by lying down. Pain in calf of short duration, may be due to co-existing deep vein thrombosis (*DVT*). Pain also can be due to ulcer/periostitis/infection. Often severity of the symptoms is not related to the severity of varicose veins. Small varicose veins may be more symptomatic than large one. In bilateral varicose veins, only one limb may be symptomatic and other limb may not. Bursting severe pain while walking may be due to deep vein thrombosis. Crampy pain during night (night cramps) is very common in these patients. Feeling of heaviness is common (**Figs 6.2A to 6.3B**).



A

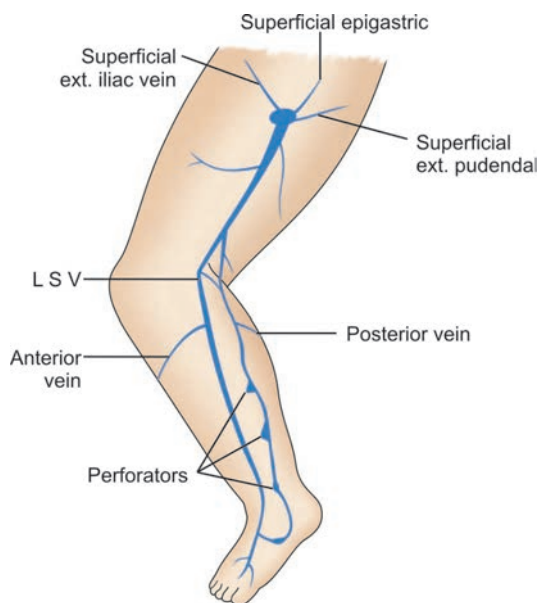


A



B

Figs 6.3A and B: Photo of short saphenous vein varicosity and its diagrammatic location.



B

Figs 6.2A and B: Long saphenous vein varicosity. Note the prominent of veins and blow outs. Note the diagrammatic representation of varicose veins.

Pigmentation

It is due to stasis and release of chemicals and usually occurs around ankle region. It is associated with itching and often ulceration.

Ulcer

History of mode of onset, duration, site of onset should be noted. Ulcer on the medial aspect of the ankle is due to long saphenous vein varicosity; on the lateral aspect is due to short saphenous vein varicosity. Discharge from ulcer—its type, smell, quantity signifies the severity of the infection. Itching and bleeding in the ulcer bed are also important to be noted.

History of trauma—Often minor trauma precipitates ulcer formation in patients with varicose vein.

History of bleeding from the vein/ulcer is an important presentation.

History of swelling around the ankle: Its duration; whether regressed any time with or without any medication; or progressive; relation to work/standing/lying down should be noted. History suggestive of difficulty/alterd gait due to pain/swelling/deformity should be noted.

History of Pain/Lump in the Abdomen

Abdominal mass/pregnancy may compress IVC/iliac veins and cause bilateral varicose veins. If compression is one side iliac veins, then varicose vein is unilateral.

History of Similar Complaints on the Other Leg

Varicose veins are often bilateral.

Past History

History suggestive of earlier deep vein thrombosis like pain, calf swelling and fever should be noted. History of immobilisation, hospitalisation; history of any previous surgery should be noted.

Treatment History

History of previous surgery for varicose vein, drug intake like warfarin for DVT, injection therapy—sclerotherapy, wearing stockings/crepe bandages should be noted.

Personal History

In females, history of varicose veins in pregnancy, and post-delivery period, use of oral contraceptive (may cause deep vein thrombosis) should be noted. History of smoking/alcohol/ working pattern should be noted

Family History

Varicose veins may be familial, which are bilateral and severe, observed in young individuals. Valves are absent/defective in these patients.

General Examination

Pulse—Rate/rhythm/character/condition of vessel wall should be noted; blood pressure is measured. Other detailed general examination is done for anaemia/oedema/jaundice/clubbing/lymphadenopathy. Raise in temperature/attitude of the patient/nutritional assessment by skin texture, subcutaneous fat, weight, body mass index/any other relevant findings should be mentioned.

Local Examination

Examination of lower limbs—symptomatic limb should be examined first.

Inspection

Examination of limbs in standing position is the first method in varicose veins (**Figs 6.4A to D**).



Figs 6.4A to D: Inspection of varicose veins should be done in standing position. Great saphenous veins on both sides should be inspected along medial aspect in standing position. Short/small saphenous vein should be inspected from behind.

Limb

Limb is looked for dilated long saphenous vein on the medial side and for short saphenous vein on posterior and lateral side. Other communicating veins are also looked for. Beginning of the varicosity in the foot, later its extent above also should be examined. Great saphenous vein tortuosity often extends into the thigh whereas short saphenous vein varicosity ends at popliteal region. Always limb is looked for skin changes, pigmentation, oedema, ankle flare, and ulcer. Extent, size, shape, floor, margin, edge, discharge in an ulcer and surrounding area, deformity should



Figs 6.5A and B: Bilateral varicose veins involving both great and small saphenous veins.



Figs 6.6A and B: Long saphenous vein varicosity. Note the prominent of veins and blow outs.

be noted. Cough impulse at saphenous opening (*Morrissey's*) may be significant (**Figs 6.5A to 6.6B**).

Swelling

In superficial varicose veins it may be a localised swelling/segmental tortuous vein. Diffuse swelling may be due to oedema/DVT.

Skin Changes

Colour changes: It may be linear redness/reddish blue colour in superficial thrombophlebitis; massive oedema with pallor and tenderness—*Phlegmasia alba dolens* (DVT of femoral vein with lymphangitis and palpable pulse); *Phlegmasia cerulea dolens* [It is cyanotic mottled skin with massive tight oedema due to occlusion of major veins (iliofemoral vein) and collaterals with absence ankle pulses] may cause venous gangrene. **Texture of the skin:** Skin may be stretched shiny, oedematous (in DVT); may also present with eczema; ulcers and scar around the ankle. Loss of hair and nail brittleness may be feature of impending venous gangrene.

Cough Impulse

Morrissey's cough impulse—Here patient is asked to cough and impulse on coughing is observed at the saphenous opening—*saphena varix*. It is done in lying down position after emptying the vein (**Fig. 6.7**).

Palpation

Ulcer, if present should be described with tenderness, induration, warmth, mobility, fixity to the underlying bone, etc.

Brodie-Trendelenburg Test (Brodie 1846; Friedrich Trendelenburg 1924)

Vein is emptied by elevating the limb and milking the vein in lying down position; a tourniquet is tied just below the saphenofemoral junction (or saphenofemoral junction can be occluded using a thumb). Saphenous opening is located 3.5 cm below and lateral to the pubic tubercle. Pubic tubercle is palpated along the adductor longus tendon which is identified by adducting the thigh against resistance. Patient is asked



Fig. 6.7: Morrissey's cough impulse.

to stand quickly. When tourniquet or thumb is released, rapid filling from above signifies saphenofemoral incompetence. This is *Trendelenburg test I*.

In *Trendelenburg test II*, vein is emptied again in lying down position and tourniquet is applied at saphenofemoral junction. After standing without releasing the tourniquet, the limb is observed. Filling of vein rapidly from below upwards can be observed within 30-60 seconds. It signifies perforator incompetence (**Figs 6.8A to 6.10D**).

Tourniquet Test for Short Saphenous Vein

Tourniquet is applied at saphenopopliteal junction after emptying the short saphenous vein by elevation. Saphenopopliteal junction is not in constant position and so it is better applied at the level of lower boundary of popliteal fossa. The tourniquet is released in standing position to look for the rapid filling from above which suggests saphenopopliteal incompetence (**Fig. 6.11**).

Three/Multiple Tourniquet Test (Oschner's Mahoner's Test) (Figs 6.12A to G)

To find out the site of incompetent perforators, three tourniquets are tied after emptying the vein-I 1. Just



Figs 6.8A to C: Emptying of the superficial varicose vein is important in all tourniquet tests for varicose veins. It is done in lying down position with elevating and milking the vein. Emptying is not done in modified Perthe's test. Note the marking of the saphenofemoral junction before applying the tourniquet is important.

below saphenofemoral junction; 2. Above knee level; 3. Another below knee level; 4. Additional tourniquets may be applied at below-knee and above ankle level. Patient is asked to stand; filling of veins and site of filling is looked for. Then tourniquets are released



Figs 6.9A to C: Note the site of applying the tourniquet at saphenofemoral junction. It is 3.5 cm below and lateral to pubic tubercle. Ideally rubber tourniquet should be used.

from below upwards to look again for incompetent perforators. Individual perforators may be tested by repeating the procedure. On standing if veins become prominent between upper most and second tourniquets, it means adductor canal perforator incompetence. Prominent veins between middle and lower signifies below knee perforator incompetence; and prominent veins below lower tourniquet, signifies incompetence of lower leg perforators.

Schwartz Test

In standing position, when lower part of the vein in leg is tapped, impulse is felt at the saphenous junction or at the upper end of the visible part of the vein. It signifies continuous column of blood and all valves between two fingers are incompetent. Positive test is usually found in gross venous varicosity (**Fig. 6.13**).

Pratt's Test

Esmarch bandage is applied to the leg from below upwards with a tourniquet tied at saphenofemoral

junction. The bandage is released later to see the 'blow outs' as perforators.

Fegan's Test (George Fegan, Dublin)

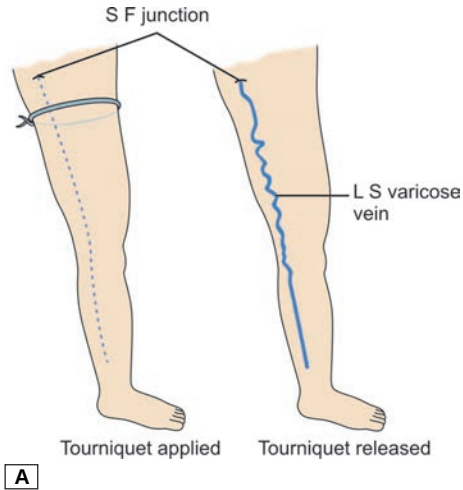
Line of varicose vein is marked. On standing, the site where the perforators enter the deep fascia bulges and these points are also marked. On lying down, button like circular depressions (crescentic gaps) in the deep fascia are felt at the marked out points which confirms the perforator site (**Figs 6.14A to C**).

Ian-Aird Test

On standing, proximal segment of long saphenous vein is emptied with two fingers. Pressure from proximal finger is released to see the rapid filling from above which confirms saphenofemoral incompetence.

Perthes Test

The affected lower limb is wrapped with elastic bandage and the patient is asked to walk around and exercise. Development of severe crampy pain in the calf signifies DVT. Test is often subjective.



Figs 6.10A to D: Tourniquet test. Tourniquet is applied after emptying the vein by elevating the leg and milking. Patient is asked to stand, tourniquet is released immediately and saphenous vein is observed. Rapid filling of vein from above signifies LSV varicosity with saphenofemoral incompetence. In test II-tourniquet applied after emptying is retained and limb is observed in standing for rapid filling of the vein from below upwards in 1 minute. It means perforators are incompetent.



Figs 6.11A to C: Small/short saphenous vein varicosity can be present along with other types like of GSV or perforators. Occasionally it can be isolated small saphenous vein varicosity. Saphenopopliteal junction is variable; but since usual site is in the lower part of the popliteal fossa, tourniquet is applied at the lower line of popliteal fossa after emptying the vein by elevation. On standing and releasing the tourniquet, rapid filling of small saphenous vein observed from above downwards signifying saphenopopliteal incompetence.

Modified Perthe's Test

Tourniquet is tied just below the saphenofemoral junction *without emptying the vein*. Patient is asked to do a *brisk walk* which precipitates *bursting pain* in the calf and also makes superficial veins more prominent. It signifies DVT.

DVT is contraindicated for any surgical intervention of superficial varicose veins. It is also contraindicated for sclerosant therapy.

Homan's Test

Homan's test is dorsiflexion of the foot to elicit pain/tenderness in the calf and *Mose's sign* is squeezing the relaxed calf muscles sideward to elicit pain/tenderness. Both tests signify *deep vein thrombosis (DVT)* (**Fig. 6.15**). Point to be remembered is that in case of acute DVT, *Homan's/Mose's tests* should be done cautiously as it will precipitate the dislodgement of the clot and embolism. On deep palpation of the calf, thickening and tenderness is felt in the calf—*Neuhof's sign*. After applying tourniquet at saphenofemoral junction, patient is asked to walk and without removing the tourniquet, limb is elevated—persisting prominent superficial veins will be observed in DVT—*Linton's test*.

Bone thickening in the shin (tibia and ankle) is important which signifies periostitis (**Fig. 6.16**).

Measurement of limb length and girth is taken especially in arteriovenous malformation with varicose veins and also to find out deformities.

Always varicose veins and perforators should be marked with a marking ink especially prior to surgery after taking consent.

Auscultation of the vein for bruit/venous hum (**Fig. 6.17**).

Examination of peripheral pulses are important (dorsalis pedis/anterior tibial/posterior tibial/popliteal/femoral).

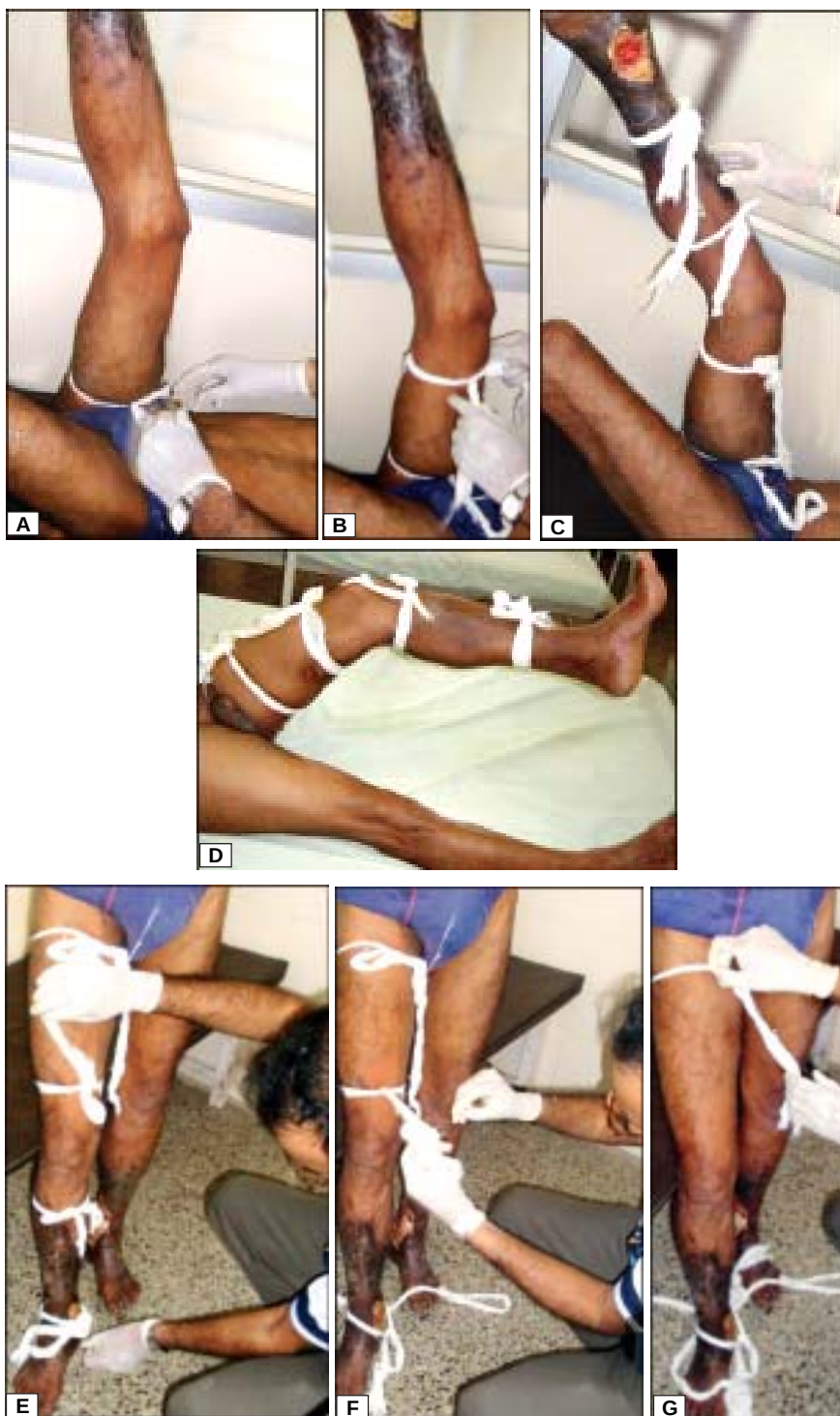
Regional lymph nodes: Vertical group of inguinal nodes and external iliac nodes (above and medial aspect of the inguinal ligament) are palpated (**Fig. 6.18**).

Ankle joint movements (plantar and dorsiflexion) are checked for any restriction. Inversion and eversion are elicited in subtalar joint.

Examination of the opposite limb both in standing and lying down position should not be forgotten.

Abdomen should be examined for any mass which might be compressing the inferior vena cava (IVC) or iliac veins causing varicose veins (**Figs 6.19 and 6.20**).

Examination of other systems should also be done (**Fig. 6.21**).



Figs 6.12A to G: Note the different phases of multiple tourniquet tests.
Ideally rubber tourniquet should be used.



Fig. 6.13: Schwartz test is done to confirm the presence of continuous column of blood.



Figs 6.14A to C: Fegan's test to find out the site of perforator. It is done both in standing and lying down position. It is done prior to surgery to have a clear idea about the site of the perforator.

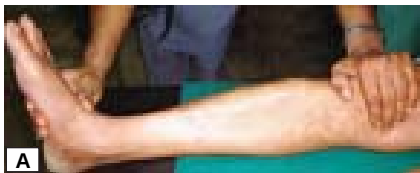


Fig. 6.15A and B: Homan's test and Mose's sign to find out acute DVT. In Homan's test foot is dorsiflexed to elicit pain/tenderness in the calf. Squeezing the relaxed calf muscles sideward to elicit pain/tenderness is Mose's sign.



Fig. 6.16: Bone thickening is checked over tibia and ankle joint using thumb. It suggests periostitis. Note also the skin changes.



Fig. 6.17: Auscultation over the groin for venous hum/bruit (of AVF) is done when needed.



Fig. 6.18: Lymph nodes in the region/groin (vertical inguinal nodes) should be examined in presence of complications like ulcer. External iliac nodes (above and medial aspect of the inguinal ligament) should also be palpated whenever required.



Fig. 6.19: Abdomen should be palpated for mass and ascites. Mass may compress the major veins causing lower limb varicose veins.



Figs 6.20A and B: Bilateral lower limb varicosity with features of IVC obstruction. Lateral and front abdominal and chest veins become prominent with flow from below upwards. (Normal flow is away from umbilicus. Below the umbilicus flow is downwards; above, it is upwards). Direction of venous flow should be confirmed using two fingers kept apart.



Fig. 6.21: Chest (cardiac and respiratory systems) should be examined. It is relevant in AV fistula/IVC obstruction.

Investigations for Varicose Veins

Laboratory Studies

No currently available lab tests are useful in the diagnosis or treatment of varicose veins. It is mainly to assess presence/absence of DVT which is absolute essential prior to intervention.

Specific Tests

Venous Doppler

With the patient standing; the Doppler probe is placed at saphenofemoral junction and later wherever required. Basically by hearing the changes in sound, venous flow, venous patency, and venous reflux can be very well identified. A uniphasic signal means flow is in one direction. A *biphasic signal* means flow in both forward and reverse direction suggesting incompetence. Reversal can often be better appreciated by releasing a tourniquet applied earlier at saphenofemoral junction.

Venous Doppler in varicose veins

To find out DVT—very important
To find out saphenofemoral, saphenopopliteal incompetence
To find out perforator incompetence
Uniphasic signals signify flow in one direction—normal;
Biphasic flow signifies reversal flow with incompetence

Duplex Scan

Duplex scan is a highly reliable U/S Doppler imaging technique (Here high resolution B mode ultrasound imaging and Doppler ultrasound is used) which along with direct visualisation of veins, gives the functional and anatomical information, and also colour map. Examination is done in standing and lying down position and also with *Valsalva manoeuvre*. Hand held Doppler probe is placed over the site and visualised for any block and reversal of flow. DVT is very well identified by this method. All patients with varicose vein should be assessed preoperatively by ultrasound for *proper venous mapping* (*Venous Haemodynamical Mapping; VHM; Cartography*), marking on the patient's limb manual and ultrasound guidance (using permanent marker). All surgeries should be done under the guidance of on table ultrasound. Postoperatively, all patients should be assessed using ultrasound for result and residual venous diseases (**Figs 6.22 to 6.24**).



Fig. 6.22: Duplex can (with Doppler machine) used for varicose veins and DVT.

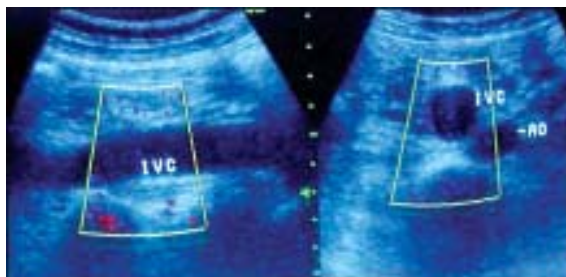


Fig. 6.23: Ultrasound showing IVC thrombosis.

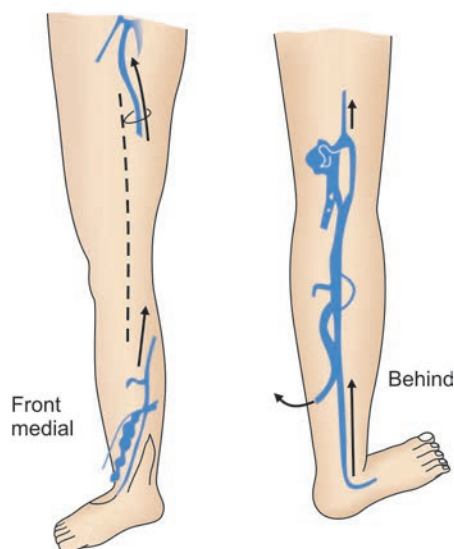


Fig. 6.24: Venous haemodynamic mapping (VHM) of the lower limb.

Venography/Phlebography

Ascending venography was a very common investigation done earlier to Doppler period.

A tourniquet is tied above the malleoli and the vein of dorsal venous arch of foot is cannulated. Water soluble dye injected, flows into the deep veins (because the applied tourniquet prevents its flow into superficial veins). X-rays are taken below and above knee level. Any block in deep veins, its extent, perforator status can be made out by this.

Note: In the presence of Duplex scan ascending venography is not a necessary investigation. If DVT is present, surgery or sclerotherapy are contraindicated.

Descending venogram is done when ascending venogram is not possible and also to visualise incompetent veins. Here contrast material is injected into the femoral vein through a cannula in standing position. X-ray pictures are taken to visualize deep veins and incompetent veins.

Phlebography

Ascending phlebography defines obstruction

Descending phlebography identifies valvular incompetence

Regularly not required to be done

Plethysmography

It is a noninvasive method which measures volume changes in the leg.

Photo plethysmography: Using probe transmission of light through the skin, venous filling of the surface venules which reflects the superficial venous pressure is measured. Initially patient performs dorsiflexion at ankle for 10 times to empty the venules and pressure tracing falls on photo plethysmography. Patient takes rest and refilling occurs. In normal people, it occurs through arterial inflow in 20-30 seconds. In venous incompetence filling also occurs by venous reflux and so refilling time is faster than normal. *Disadvantage:* Site of reflux cannot be localised by this method.

Air plethysmography: Patient is initially in supine position with veins emptied by elevation of leg. Air filled plastic pressure bladder is placed on calf to detect volume changes. Minimum volume is recorded. Patient turned to upright position and venous volume is assessed. Maximum venous volume divided by time required to achieve maximum venous volume gives the *venous filling index (VFI)*. VFI is a measure of reflux. *Ejection fraction* is volume change measured prior and after single tip toe manoeuvre which is a measure of calf pump action. *Residual venous fraction* is an index of overall venous function which is venous volume in the leg after 10 toetip manoeuvres divided by venous volume prior to manoeuvre. *A patient with increased VFT and diminished ejection fraction will benefit from surgery.*

Ambulatory Venous Pressure (AVP)

It is an invasive method. Needle inserted into dorsal vein of foot is connected to transducer to get its pressure

which is equivalent to pressure in the deep veins of the calf. Ten tiptoe manoeuvres are done by the patient. With initial rise in pressure, pressure decreases and eventually stabilizes with a balance. Pressure now is called as *ambulatory venous pressure (AVP)*. After stopping exercise, veins are allowed to refill with return of pressure to baseline. Time required for pressure to return to 90% of baseline is called as *venous refilling time (VRT)*. Raise in AVP signifies venous hypertension. Patients with AVP more than 80 mm Hg has 80% chances of venous ulcer formation.

Varicography

Here non-ionic, iso-osmolar, non-thrombogenic contrast is injected directly into the variceal vein to get a detailed anatomical mapping of the varicose veins. It is used in recurrent varicose veins or with anatomical variations.

Arm-Foot Venous Pressure

Foot pressure is not more than 4 mm Hg above the arm pressure. Foot venous pressure will be as high as 10-15 mm Hg above of hand venous pressure—*Raju test*.

Patients with varicose veins may have spuriously **positive D-dimer test** result because of chronic low level thrombosis within varices.

Muscle pump ejection fraction assessment may be useful to demonstrate reflux.

U/S abdomen, peripheral smear, platelet count, and other relevant investigations are done depending on the cause of the varicose veins.

If venous ulcer is present, then the discharge is collected for culture and sensitivity, biopsy from ulcer edge is taken to rule out Marjolin's ulcer, plain X-ray of the part is taken to find out periostitis (**Figs 6.25A and B**).

Radioactive Fibrinogen Test

Sodium iodide 100 mg orally is given to the patient 24 hours before the test to block the thyroid activity. I^{125} labelled fibrinogen 100 μ curies is injected intravenously. First radioactivity of heart is measured by placing the scintillation counter over the precordium. Reading obtained by this is adjusted as 100%. After



Figs 6.25A and B: Venous ulcer showing periostitis features in the X-ray taken.

that legs are elevated using adjustable stands and to prevent venous pooling, scintillation counter is placed over the calf. Counting in the leg is done from below upwards at 5 cm intervals. Procedure is done in preoperative period; on 1st, 3rd and 6th postoperative days. A 20% or more rise in percentage value suggests deep vein thrombosis in leg. I^{125} labelled fibrinogen is used (earlier I^{131} labelled fibrinogen was used) because it has got softer radioaction and its detectability is possible with much lighter and mobile apparatus.

Routine Investigations

Haematocrit, blood urea, serum creatinine, blood sugar; Chest X-ray, ECG. It is mainly done to prepare the patient for surgery-for anaesthesia purpose.

Surgical Anatomy of Lower Limb Veins

Superficial venous system: It is located in the saphenous compartment which is between subcutaneous plane and deeper aponeurotic plane. GSV and SSV are in this plane.

Great (Long) saphenous vein (GSV)—It runs from the medial end of the dorsal venous arch up along the anteromedial aspect of the leg and thigh until it empties into the femoral vein. There is one valve at this junction—is called as ostial/terminal valve and reflux through it is called as *ostial reflux*. There is one more valve proximal to the main junctional tributary veins and is called as preterminal valve. More than 50% reflux occurs at preterminal valve (*preterminal reflux*).

Small (Short) saphenous vein (SSV)—It runs from the lateral end of the dorsal venous arch up along the posterolateral aspect of the calf until it passes through the popliteal fossa behind the knee and empties into the popliteal vein just above the knee. *Tributaries* are located in the subcutaneous plane which joins the saphenous system. Superficial circumflex vein (often joins AAGSV), superficial external pudendal vein, superficial inferior epigastric vein, anterior vein of the leg, posterior arch vein of the leg (joins GSV), posterolateral venous chains of leg—are different tributaries. Long intersaphenous communicating vein often exists between cranial extension of SSV to join GSV and can be varicose and pathological and is called as *communicating vein of Giacomini-Cruveilhier*. **Anterior accessory great saphenous vein (AAGSV, Anterolateral vein of thigh)** is communicating vein into the GSV anteriorly and laterally. AAGSV communicates into GSV usually just proximal to preterminal valve (60%); often at confluence (39%); rarely onto femoral vein (1%). In many patients with varicose veins it is this vein which is diseased than GSV. It often receives superficial circumflex vein before joining the GSV.

Perforator system of veins (communicating veins)—They pass through the deep fascia carrying blood from superficial to the deep system. All blood flow is one way maintained through valves. Incompetence of these valves leads to appearance of superficial varicosity during exercise.

Deep venous system: It comprises femoral and popliteal veins; pairs of venae comitantes of the tibial,

posterior tibial and peroneal arteries (total 6 venae comitantes, soleal and gastrocnemius veins join to form popliteal vein); valveless blood lakes within the calf muscles communicates with superficial system via saphenofemoral junction, mid-thigh perforators, short-saphenopopliteal junction and calf perforators.

The calf muscle pump: It is often referred to as “*peripheral heart*”. Its inflow into a segment of deep vein is through intake valves of the perforating veins and segment of deep vein below. Its outflow is through outflow valve to the deep vein segment above. It has got soleal veins (flush up the blood) and gastrocnemial veins (Gilot) (push up the blood) to have effective motor venous return (**Figs 6.26A to D**).

Different Types of Perforators

Para Achilleal (Bassi); Ankle perforators: (*May or Kuster*); **Lower leg perforators** between deep veins and posterior arch vein (*Cockett*): I (posteroinferior to medial malleolus), II (10 cm above the medial malleolus), III (15 cm above medial malleolus); **Gastrocnemius perforators** between GSV and deep veins—upper proximal paratibial (*Boyd*)—below knee; Lower and medial paratibial (*Sherman*); ‘24’ *cm perforator* between deep veins and GSV; **Mid-thigh perforator** between deep vein and GSV (*Dodd*); *Hach perforator in the posterior thigh*; **Hunter’s adductor canal perforator** in the thigh (**Fig. 6.27**).

Physiology of Venous Blood Flow in Lower Limb

Veins are thin walled vessels with collapsible walls that assume an elliptical configuration in collapsed state and circular configuration in the filled state. Venous valves are abundant in the distal lower extremity and the number of valves decreases proximally, with no valves in superior and inferior vena cava.

Factors affecting venous return are:

- Arterial pressure across the capillary increases the pumping action of vein;
- Calf musculovenous pump:** During contraction phase of walking, pressure in the calf muscles increases to 200-300 mm Hg; This pumps the blood towards the heart; During relaxation phase of walking, pressure in the calf falls and so it

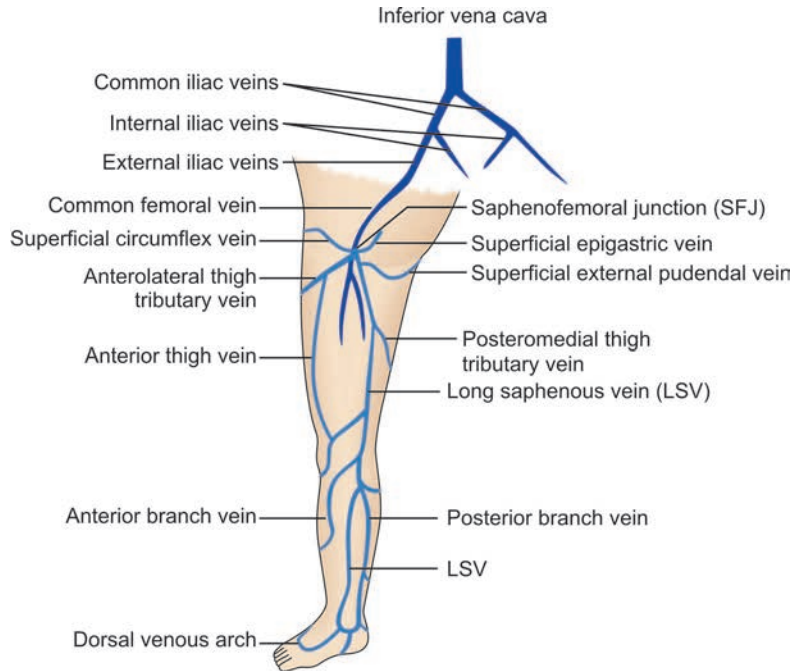


Fig. 6.26A

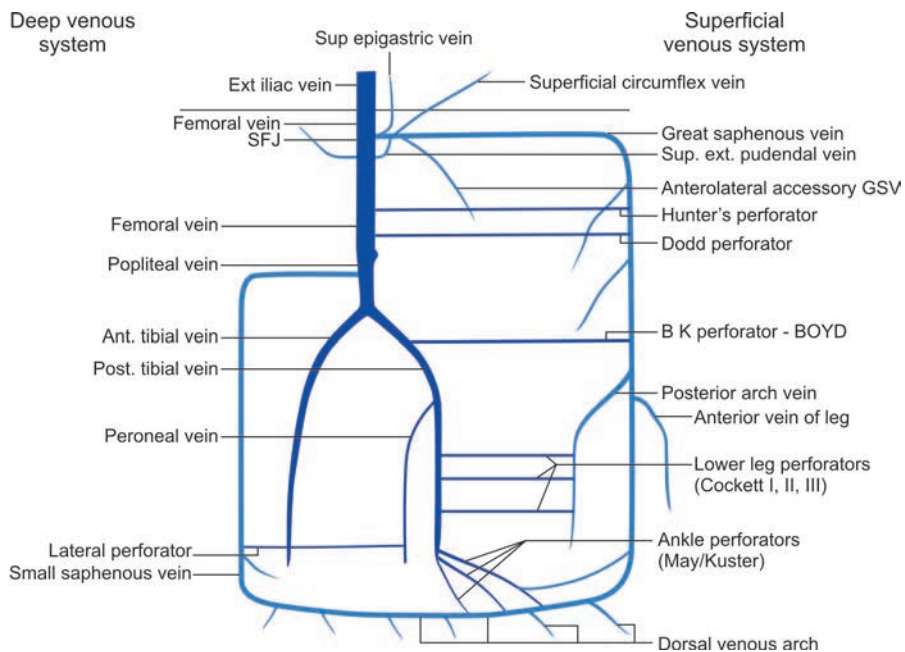


Fig. 6.26B

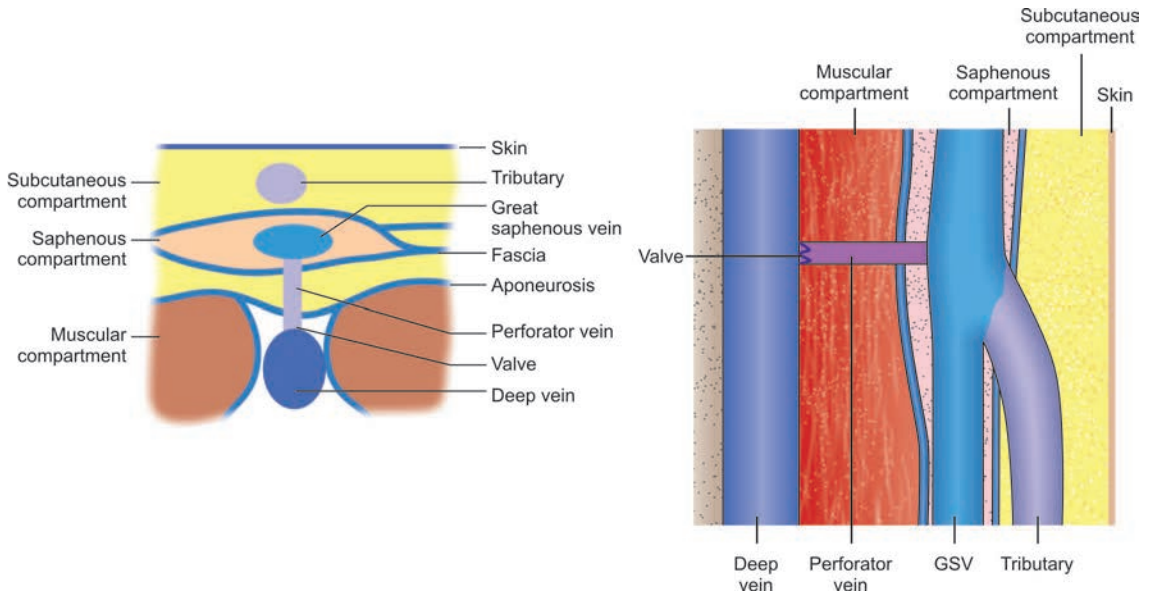


Fig. 6.26C

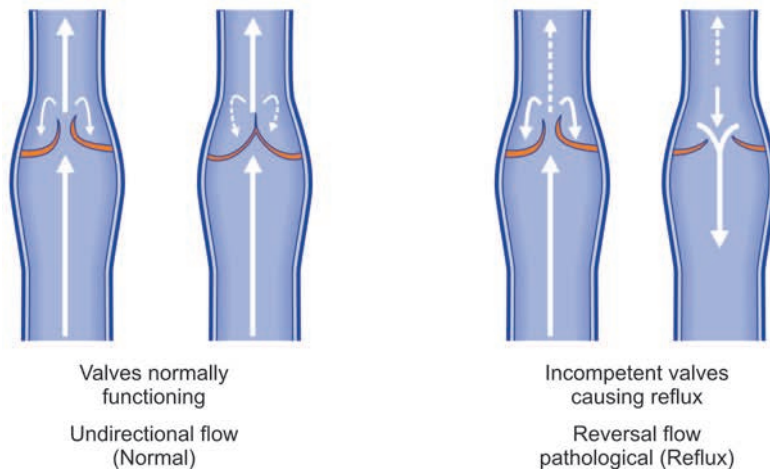


Fig. 6.26D

Figs 6.26A to D: Diagrams showing venous anatomy of the lower limb. Also note the functioning of the valve in the vein.

allows blood to flow from superficial to deep veins through perforators; Normally while walking, pressure in the superficial system is 20-30 mm Hg at the level of ankle; During walking, foot pump mechanism propels blood from plantar veins into the leg;

c. *Gravity:* Pressure in the vena cava where it enters the right atrium is very low (-5 mm Hg) which allows rapid filling of vena cava from high pressure peripheral veins.

Factors responsible for venous return: Negative pressure in thorax; Peripheral pump—calf muscle;

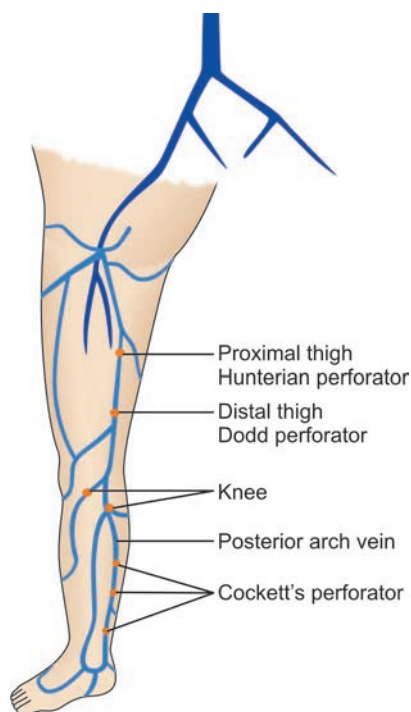


Fig. 6.27: Diagram showing different perforators.

Vis-a-tergo of adjoining muscle; Nonrefluxing valves in course of veins.

Venous Pathology

Venous pathology develops when venous return is impaired. It can be of *deep, superficial or mixed types*. It can be due to primary muscle pump failure, venous obstruction, and venous valvular incompetence. It may be segmental or affecting the entire leg. Here venous wall shows increase in collagen and reduction in elastin.

Causes of primary muscle pump failure are—muscle wasting, neuromuscular disease, deep fasciotomies, local vein valve failure. It leads to nonreduction of postambulatory pressure; causes increased hydrostatic pressure and thus decreased arterial inflow. Present concept is inflammatory pathology of the venous wall and valve. Bottom to top theory is newly changed present concept with segmental pathophysiology. So VHM (venous mapping) is essential prior to surgical intervention. Incompetence can be of three types—perforator; superficial and deep.

Perforator incompetence is reversal of normal flow across the perforators. Normal flow is from superficial veins to deep veins. Perforator valve failure causes deep to superficial reflux causing congestion and venous hypertension. It causes transmission of extreme high pressure (150-200 mm Hg) generated at the deep veins into the superficial veins. This high pressure reflux (*hydrodynamic reflux*) causes distal valvular incompetence of the superficial venous system causing varicosity and its problems. Problems here are more rapid and progressive than just superficial vein incompetence.

Superficial vein incompetence is the most common form of the venous disease. It shows retrograde flow (*hydrostatic reflux*) due to malfunctioning valves. It is the tributaries which become commonly incompetent and diseased as it is in subcutaneous plane with less support and also has got thin muscle in the wall. Here effects are slow and gradual.

Deep incompetence may be isolated incompetence of deep veins or may be associated with incompetence of superficial venous system. Here blood pumped out in normal volumes but extremity refill includes normal arterial inflow and also pathological reflux. Venous refill is rapid with normal or elevated ambulatory venous pressure (**Fig. 6.28**).

Varicose Veins

Definition of the Varicose Veins

It is *dilated, tortuous and elongated* superficial vein with reversal of blood flow due to incompetence of valves. It is seen only in human beings due to erect posture. It is not seen in animals. A varicose vein is one which has *permanently* lost its valvular efficiency. As a result of continuous dilatation under pressure in course of time, varicose vein becomes elongated, tortuous, pouched, thickened, inelastic and friable structure. Incidence of varicose veins is 5% in general population.

Presentations of Varicose Veins

It is more common in females (10:1). It is much more common in females with a family history. Often it is familial. *Familial varicose veins* begin in younger age group and are seen bilaterally, involve all veins including deep veins. Presents with visible dilated veins

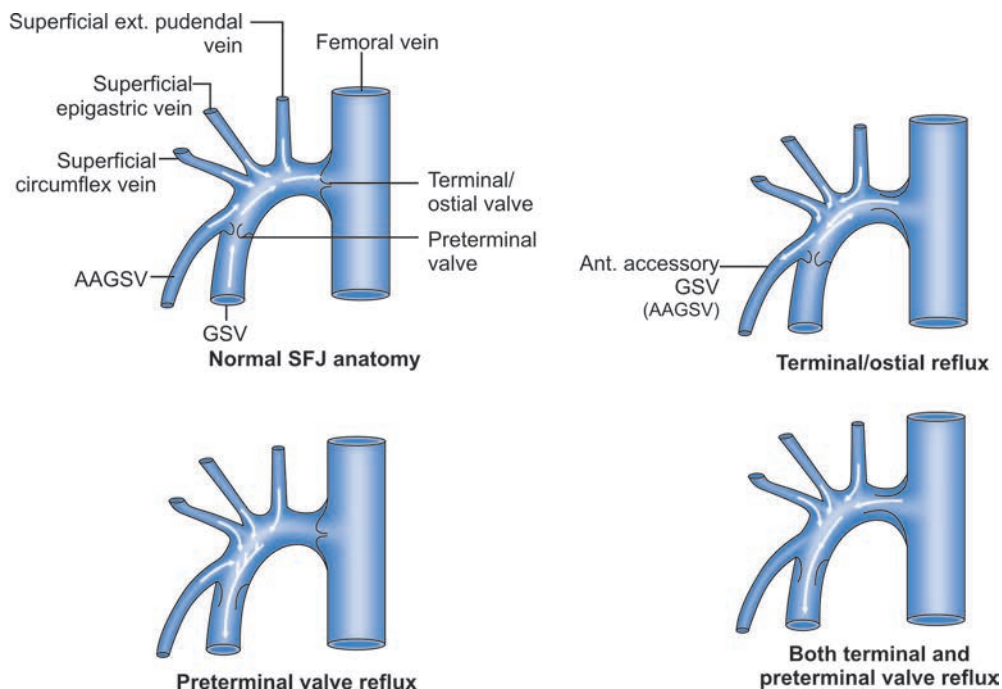
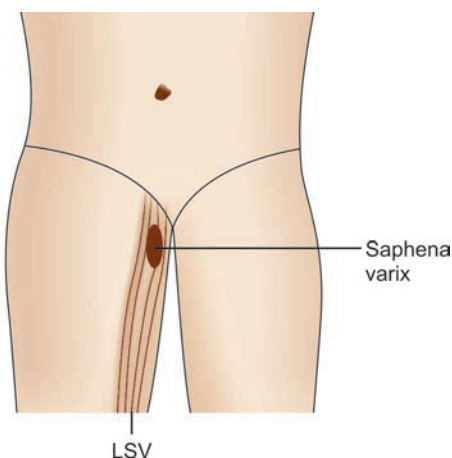


Fig. 6.28: Great saphenous vein anatomy at its saphenofemoral junction showing reflux.

in the leg with pain, distress, *nocturnal cramps*, feeling of heaviness, muscle fatigue, throbbing heavy legs, (restless legs), soreness, burning, pruritus. Often there is pedal oedema, pigmentation, dermatitis, ulceration, tenderness, restricted ankle joint movement, bleeding, and positive cough impulse at the sapheno-femoral junction. Thickening of tibia occurs due to

periostitis. It may present with DVT, especially in pregnancy. Local gigantism may be the presentation in varicose veins due to congenital AV malformation (Figs 6.29A and B).

Note: Extent of valvular incompetence is not related to the presence and severity of the symptoms.



Figs 6.29A and B: Saphena varix in the groin—near the saphenous opening.

Symptoms in varicose veins

Dilated tortuous vein – asymptomatic but cosmetic
 Dragging pain
 Heaviness/tiredness in the legs
 Night time cramps – usually late night - typical
 Oedema feet/itching/thickening feet/eczema feet and leg
 Discolouration/ulceration in the feet/painful walk
 Bleeding blow outs

Signs

All different positive tests
 Blow outs – localised dilated vein segment suggests incompetent perforator – Fegan's test
 Superficial thrombophlebitis
 Ankle flare (**Fig. 6.30**)
 Dermal flare (thread veins) < 1 mm – it is within the skin
 Reticular veins (1-3 mm) in the subcutaneous tissue
Saphena varix – A large varicosity in the groin (of GSV; often of anterolateral thigh vein)
 Talipes equino varus
Champagne bottle sign (inverted beer bottle look) – contraction of ankle skin and subcutaneous tissue with prominent oedematous calf.

Causes of pain/cramps in varicose veins/venous diseases

Increased venous wall tension – chronic venous hypertension
 Hypoxia of tunica media of the venous wall due to altered function of vasa vasorum
 Increased capillary pressure
 Hyperviscosity of red cells
 Platelet hyperaggregation
 Reduction in capillary permeability causing *capillary functional disorder*
 Altered cutaneous microcirculation due to leucocyte adhesion and accumulation into the venous wall; release of free radicals cause *microvascular lesional disease*.

Oedema in venous diseases

Can be localised or generalised
 Localised oedema is due to ankle flare or dilatation of medial marginal vein
 Cellulitis and lymphangitis association causes oedema, scarring and thickening of dermal and subdermal tissues – *lipodermatosclerosis* (brawny induration)
 Ankle becomes narrower due to contraction of skin and subcutaneous tissues but calf remains prominent – *champagne bottle appearance* (**Fig. 6.31**)
 Pale atrophic skin with white patches surrounded by dilated capillaries and pigmentation – *atrophic blanche*.

Aetiologies for Varicose Veins

Varicosities are more common in lower limb. Because of erect posture long column of blood has to be supported which can lead to weakness and incompetence of valves leading to varicosities.



Fig. 6.30: Typical ankle flare. Thread veins are dermal and are < 1 mm in size. Reticular veins are subcutaneous and are between 1-3 mm in size.

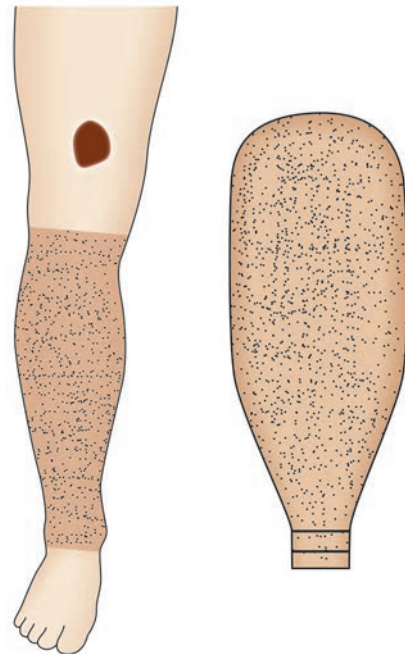


Fig. 6.31 *Champagne bottle sign/inverted beer bottle sign* is seen in lipodermatosclerosis due to prominent calf with narrow ankle contracted skin and subcutaneous tissue. Sign is often observed in DVT also.

Primary varicosities are due to—

Congenital incompetence or absence of valves; weakness of valves; weakness or wasting of muscles; stretching of deep fascia. It is precipitated by prolonged standing and recurrent thrombophlebitis.

Secondary varicosities are due to—

Recurrent thrombophlebitis; occupational—standing for long hours; obstruction to venous return like abdominal tumour, retroperitoneal fibrosis, pelvic mass; ascites; lymphadenopathy; pregnancy (due to progesterone hormone); acquired AV fistula (due to surgery/trauma). It may be due to previous deep venous thrombosis.

Congenital

Congenital A-V malformations; Klippel-Trenaunay syndrome; Avalvulia.

Sites where varicosities can occur

Lower limb
Pampiniform plexus of veins—varicocele
Vulval/ovarian varices
Sites of portosystemic anastomosis (Piles/oesophageal varices/gastric varices)

Types of Varicose Veins

1. Long saphenous vein varicosity.
2. Short saphenous vein varicosity.
3. Varicose veins due to perforator incompetence.
4. Thread veins (dermal flares): Are small varices in the skin usually around ankle which look like dilated, red or purple network of veins of < 1 mm in size.
5. Reticular varices: Are slightly larger than thread veins located in subcutaneous region 1-3 mm in size.
6. Combinations of any of above.

It can be—Primary varicose veins; Secondary varicose veins; Reticular veins (Venulectasia); Telangiectasias (Spider veins, Hyphen webs, Thread veins). *Corona phlebectatica* are blue telangiectasias on the medial aspect of the foot below the malleolus around ankle level. More than 5 such lesions are the best independent predictor of the skin changes.

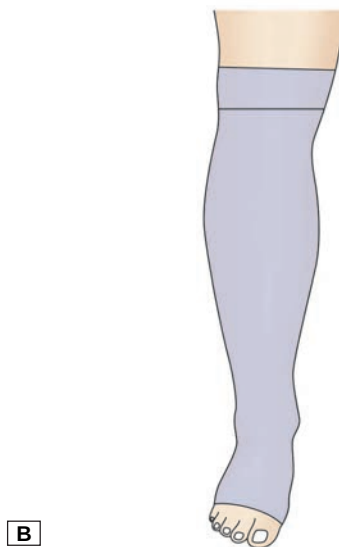
Venous disability scoring system

Score 0	Asymptomatic
Score 1	Symptomatic but able to carry out activities without any therapy
Score 2	Symptomatic – can do activities only with compression/limb elevation
Score 3	Symptomatic – unable to do daily activities even with compression or limb elevation

Venous segmental disease score (venous clinical scoring system) is done based on different symptoms/signs/ulcer activity/compression therapy with 10 parameters with each having 3 scores as mild/moderate/severe (**Figs 6.32A and B**).

Complications of varicose veins

Haemorrhage: Venous haemorrhage can occur from the ruptured varicose veins or sloughed varicose veins, often torrential, but can be controlled very well by elevation and pressure bandage
Eczema and dermatitis
Periostitis causing thickening of periosteum
Venous ulcer
Marjolin's ulcer – due to *unstable scar of long duration*—very well differentiated squamous cell carcinoma
Lipodermatosclerosis
Ankylosis of the ankle joint
Talipes equino varus
Deep venous thrombosis
Calcification



Figs 6.32A and B: Crepe bandage or stockings are used in varicose veins or in DVT. After any intervention for varicose veins, crepe bandages/stockings should be applied for 6 months. It should be worn from toes to knee joint.

Lipodermatosclerosis and development of different problems in varicose veins

Fibrin deposition, scarring and tissue hypoxia due to chronic venous hypertension around ankle joint is called as *lipodermatosclerosis*. It is irreversible change in the soft tissue which eventually leads into ulceration (**Fig. 6.33**).

Two theories: Fibrin cuff theory; White cell trapping theory

Incompetence of venous valves → stasis of blood → chronic ambulatory venous hypertension (Pressure up to 80-100 mm Hg) → defective microcirculation → RBC's diffuses into tissue planes → lysis of RBC's → release of haemosiderin, pigmentation → dermatitis → capillary endothelial damage → prevention of diffusion and exchange of nutrients → severe *anoxia* → chronic venous ulceration (**Fibrin cuff theory**).

Inappropriate activation of trapped leucocytes release proteolytic enzymes which cause cell destruction and ulceration—**White cell trapping theory**.

Venous Ulcer

It is the complication of varicose veins or deep vein thrombosis.

Pathogenesis of Venous Ulcer

Varicose veins or DVT which are recanalised, eventually causes chronic venous hypertension around ankle → causes hemosiderin deposition in the subcutaneous plane from lysed RBC's, Eczema → dermatitis and lipodermatosclerosis → fibrosis → anoxia → **ulceration**. Area where venous ulcer commonly develops is around and above the medial malleoli because of presence of large number of perforators which transmit pressure changes directly into superficial system. This area is called as **Gaiter's zone** (**Fig. 6.34**). It can be seen on both malleoli. Ulcer is often large, nonhealing, tender, recurrent with secondary infection. Vertical group of inguinal lymph nodes are usually enlarged and tender. Often it leads to scarring, ankylosis, *Marjolin's ulcer* formation. Sloughing from the ulcer bed may give way causing venous haemorrhage. *Periostitis* is common which also prevents ulcer from healing. Due to regular walking on toes to get relief from pain, causes contraction and extra articular fibrosis of Achilles tendon—*talipes equino varus* (**Figs 6.35A to C**).



Fig. 6.33: Lipodermatosclerosis left leg.

CEAP Classification (Advanced 2004)

It is the classification used for lower extremity venous diseases.

CEAP Classification

- C**— Clinical signs (grade 0-6); (A) for asymptomatic or (S) for symptomatic presentation
- E**— Aetiological classification (congenital, primary, secondary, no venous aetiology)
- A**— Anatomic distribution (Superficial (As), deep (Ad) or perforator (Ap))
- P**— Pathophysiologic dysfunction (reflux or obstructive or both or no pathophysiologic dysfunction)

Grading of clinical signs (C)

- 0— No visible or palpable signs of venous diseases
- 1— Telangiectases, reticular veins or malleolar flare
- 2— Varicose veins
- 3— Oedema without skin changes
- 4— Skin changes due to venous diseases like pigmentation, eczema or lipodermatosclerosis 4a— pigmentation; 4b—lipodermatosclerosis, atrophie blanche
- 5— Skin changes as above with healed ulceration
- 6— Skin changes as above with active ulceration

Anatomical distribution (A)

As—superficial system

- 1. Telangiectases, reticular veins
- 2. Great saphenous vein above the knee—ostial and preterminal
- 3. Great saphenous vein below the knee
- 4. Small saphenous vein
- 5. Nonsaphenous—43%

Ad—deep system

From 6 to 15

Ap—perforator system

- 17—Perforator vein (PV) of the thigh
- 18—Perforator vein (PV) of the calf and leg

An—no anatomical lesion identified

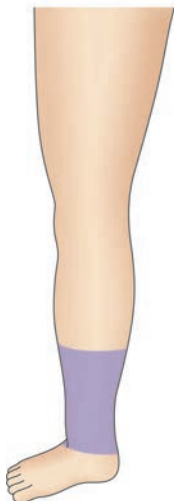


Fig. 6.34: Gaiter's zone is the handbreadth area around ankle where problems/ complications/ulceration of venous disease occur.

Note:

50% of venous ulcer occurs as a result of recanalisation of DVT, and the leg is commonly called as *post-phlebitic limb (leg)*. It presents with all complications of venous diseases like eczema, ulceration, lipodermatosclerosis, and venous ulcers. Here surgery for superficial varicose veins is contraindicated. Most of the venous ulcers have surrounding lipodermatosclerosis. *Lipodermatosclerosis is due to pigmentation, thickening, chronic inflammation and induration of the skin in calf and around ankle.* 70-80% of leg ulcers are venous ulcers.

Complications of venous ulcers

- Haemorrhage
- Marjolin's ulcer
- Infection
- Talipes equino varus
- Periostitis is common over the tibia/calcaneum/other
- Foot bones
- Disability
- Calcification
- DVT

Deep Vein Thrombosis (DVT)

It is thrombosis of the deep venous system. It can be *acute* or *recurrent*. It can be *occlusive* or *non-occlusive*. It can be *free* thrombus or *fixed* thrombus.



A



B



C

Figs 6.35A to C: Typical site of venous ulcer. Often venous ulcer can be bilateral.

It can be *propagative* which propagates proximally and has higher chance of formation of embolism or *nonpropagative*. Factors— *Virchow's triad (1856)*: Stasis; Hypercoagulability; Vein wall injury.

Causes

Following *childbirth*; trauma; muscular violence; *prolonged immobility*; debilitating illness, obesity, bed rest, pregnancy, puerperium, oral contraceptives, and oestrogens.

Postoperative thrombosis: Common after the age of 40 years. Incidence following surgeries is 30%. In 30% of cases both legs are affected. Usually it is seen after prostate surgery, hip surgery, major abdominal surgeries, gynaecological surgeries, cancer surgeries. Bedridden for *more than 3 days* in the postoperative period increases the risk of DVT.

Spontaneous thrombosis is common in visceral neoplasm like carcinoma pancreas or carcinoma stomach. It is often migrating type.

Thrombus may start in a venous tributary which may eventually extend into the main vein causing DVT.

Axillary vein thrombosis: Upper limb DVT (5% of total DVT) can occur spontaneously, following compression by cervical rib, by various causes of thoracic inlet syndrome, or arm being in the hyperabduction state for prolonged period (e.g painting the ceiling, athletes, swimmers), after axillary lymph node block dissection, after radiotherapy to axilla, occasionally as a complication of venous cannulation. Idiopathic upper limb DVT may be due to some occult malignancy in the body. Even though upper limb DVT is less common chances of pulmonary embolism is more—33% of upper limb DVT can lead into pulmonary embolism.

Polycythaemia vera, thrombocytosis; deficiencies of antithrombin III, protein C, protein S; factor V of Leiden, antiphospholipid syndrome, thrombophilia, recent myocardial infarction, heart failure, nephrotic syndrome, *thrombosis* (in people who sit on computer for long time) are other causes.

Sites: (1) Pelvic veins—Common. (2) Leg veins—Common in femoral and popliteal veins (Common on left side). (3) Upper limb veins—Not uncommon (Axillary vein thrombosis).

Phlegmasia alba dolens: It is DVT of *femoral vein* (deep femoral vein commonly) causing painful congestion and oedema of leg, with lymphangitis, which further increases the oedema and worsens the situation (*White leg*). **Phlegmasia cerulea dolens:** It is extensive DVT of *iliac and pelvic veins* causing *blue leg* with either venous gangrene or areas of infarction (**Fig. 6.36**).

Clinical Features

Fever is the earliest and common symptom. Pain and swelling in the calf and thigh, commonly associated



Fig. 6.36: Venous gangrene right leg.

with fever. Pain is often so severe that the patient finds difficult to flex or move the leg. Leg is tense, tender, warm, pale or bluish with stretched and shiny skin (**Fig. 6.37**).



Fig. 6.37: DVT both legs.

Positive Homan's sign: Passive forceful dorsiflexion of the foot with extended knee will cause tenderness in the calf. Positive Homan's sign is confirmative sign of DVT; but absence of Homan's sign is not a reliable indicator of absence of DVT.

Mose's sign: Gentle squeezing of lower part of the calf from side-to-side is painful. Gentleness is very important otherwise it may dislodge a thrombus to form an embolus.

Neuhof's sign: Thickening and deep tenderness is elicited while palpating deep in calf muscles.

Linton's test: After applying proximal tourniquet: with elevation after walk; superficial veins are still prominent. Most often, DVT is *asymptomatic* (60%) and

presents suddenly with features of pulmonary embolism like chest pain, breathlessness and haemoptysis.

Investigations

Venous Doppler; Duplex scanning (**Fig. 6.38**).

Venogram—It is invasive method. It is good test to find out occlusive and nonocclusive thrombus, but has got 10% interpretation error. Ascending functional venogram is better.

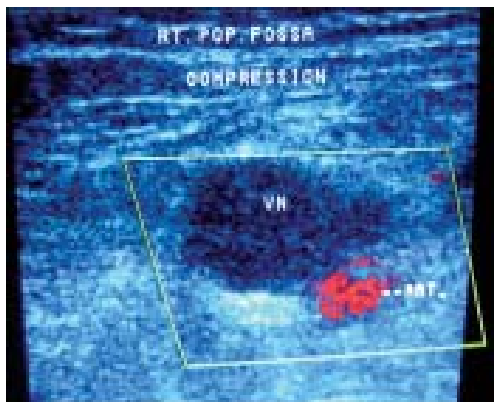


Fig. 6.38: Duplex scan showing DVT in leg. Deep vein thrombosis is contraindicated for varicose vein surgery.

Radioactive I ¹²⁵ fibrinogen study; haemogram with platelet count.

Impedance plethysmography is not commonly done procedure. When applied cuff in the upper thigh is deflated, there is normally rapid outflow and reduction of volume. In venous thrombosis, outflow wave is prolonged.

Fibrin/fibrinogen assays: It is the measurement of degradation of intravascular fibrin. In *D-dimer test* cross linked degradation products are measured. Negative D-dimer test has high negative predictive value (98%) which is more reliable than positive D-dimer test.

MR venography is useful in imaging iliac veins and IVC.

Prevention of DVT

Care has to be taken to see for proper positioning of legs with no pressure on the calf muscles. Pressure bandage/stockings to the legs have to be applied during major surgeries, and laparoscopic surgeries. During postoperative period, elevation, massaging, pressure

bandage, early ambulation, maintaining hydration are essential measures. Low dose heparin is given in suspected cases, in major surgeries and continued during postoperative period till the patient is ambulated. 5000 units is given subcutaneously 2 hours before surgery. Various measures like graduated static compression, elastic stockings, electrical stimulation of calf muscles, pneumatic compression are used to prevent sluggish flow of blood. Intravenous 500 ml dextran-70 during surgery and another 500 ml in post-operative period in 24 hours can also be used to prevent DVT.

Effects and sequelae of DVT

- Pulmonary embolism—15%
- Infection
- Venous gangrene
- Partial recanalisation, chronic venous hypertension around the ankle region causing venous ulcers—chronic venous insufficiency (CVI)
- Recurrent DVT
- Propagation of thrombus proximally

Superficial Thrombophlebitis

It is thrombosis with inflammation of superficial veins (**Figs 6.39A and B**). It can be acute—due to IV cannulation, trauma, minor injury/infection, hypercoagulability; spontaneous—due to polycythaemia, polyarteritis nodosa, TAO; migratory thrombophlebitis (*Trousseau's sign*—1876—Trousseau himself had migratory thrombophlebitis due to advanced carcinoma) is due to underlying gastrointestinal malignancy commonly carcinoma pancreas. *Mondor's disease* is superficial thrombophlebitis of subcutaneous veins of breast and chest wall. Clinical features are pain, occasionally fever, redness, tenderness, and cord-like thickening of veins. *Complications are*—DVT, venous valve destruction and incompetence, infection like cellulitis, embolism. It is managed by anti-inflammatory drugs, pressure bandage, antibiotics.



Figs 6.39A and B: Superficial thrombophlebitis forearm and arm.

7

Examination of Lymphatic System

Surgical Anatomy

Primordial lymphatic system begins to develop during 6th week of development adjacent to jugular vein as lymph sacs. Peripheral lymphatic systems develop from these primordial lymph sacs. Lymphatic system has three components. *Terminal lymphatic capillaries*, which have high porosity absorb lymph, macromolecules, cells and microbes from tissues into the system; *lymphatic vessels* which collect and transport lymph; *lymph nodes* which are interposed in the lymphatic pathway filters lymph and maintain immunity of the body. Lymphatic vessels run adjacent to main blood vessels reaching the major lymphatic channels. *Cisterna chyli* is formed in the abdomen, continues as thoracic duct (formed at 9th week of gestation) in the thorax which has got initial main course towards right side of the mediastinum; but later migrates towards left side entering the internal jugular vein at its joining point of the subclavian vein. In the periphery there is hardly any lymphovenous communications. Lymphovenous communications occur at lymph node level; iliac, subclavian and jugular levels. Lymphatics are absent in epidermis, cornea, CNS, cartilage, tendon and muscle.

Great lymph ducts are the thoracic duct—single; right lymph duct—single; subclavian, bronchomediastinal and jugular trunks on both sides. These ducts contain valves to prevent backflow.

Cisterna chyli is formed by joining of right and left lumbar lymphatic trunks and intestinal lymphatic duct. Lumbar trunks are short lymph vessels arising from para-aortic lymph glands. It receives lymph from lower limb, pelvis and pelvic viscera, kidney, adrenal and abdominal wall deep lymphatics. Left lumbar trunk is behind the aorta. Intestinal lymph duct arises from preaortic nodes. It joins the cisterna chyli from front. It receives lymph from stomach, intestines, liver

(except most convex surface which drains into right lymph duct), spleen and pancreas. Cisterna chyli is a lymph sac lying in front of the L₁ and L₂ vertebrae between aorta and crus of the diaphragm. From its upper end it continues as thoracic duct. *Thoracic duct* passes through the aortic orifice of the diaphragm, runs medial to azygos vein and right of the aorta in posterior mediastinum. In front it is related to oesophagus, diaphragm and pericardium; behind right intercostals arteries, hemiazygos and accessory hemiazygos vein. At the level of 7th thoracic vertebra it crosses towards left side behind the oesophagus obliquely reaching left side at 5th thoracic vertebral level. It runs upwards between left margin of oesophagus, medial part of left pleura, and behind left subclavian artery. In the neck it passes in front of vertebral system (vertebral vessels and sympathetic chain) and behind carotid system (Common carotid artery, internal jugular vein, vagus nerve), crossing scalenus anterior, phrenic nerve and transverse cervical and suprascapular arteries ending as a single vessel at the junction of internal jugular vein and subclavian vein with a valve. Tributaries of thoracic duct are: trunk from lateral intercostals nodes from lower six spaces; efferents from posterior mediastinal nodes, lateral intercostal nodes of upper six spaces, left jugular lymph trunk from head and neck region, left subclavian lymph trunk from left upper limb, left bronchomediastinal trunk from left side of the thorax (**Fig. 7.1**). Single termination of duct is common (77%); but double/triple/quadruple terminations are known to occur. Occasionally it may end in left subclavian vein, left vertebral vein, right internal jugular vein, right subclavian vein. Thoracic duct is 45 cm in length and 5 mm wide (wider at both ends; narrow in the middle).

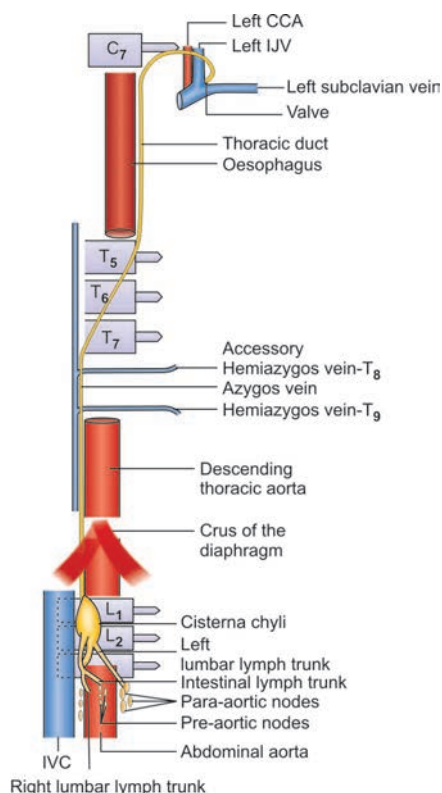


Fig. 7.1: Anatomy of thoracic duct; cisterna chyli; tributaries of thoracic duct.

Right lymph duct is 2.5 cm in length, formed by right jugular, right subclavian and right broncho-mediastinal trunks; runs on the scalenus anterior joining the junction of right internal jugular vein and subclavian vein.

There are about total 450-600 lymph nodes in the body. Around 200 in the neck; around 100 in the thorax; around 50 - 60 in the axilla; around 250 in the abdomen and pelvis; around 50 in the groin area.

Lymphatic Watersheds of Skin

Lymph from the dermis and appendages drain into a plexus in deep fascia which in turn drains into respective lymph nodes. There are six watershed areas in the body for lymphatic drainage. One vertical midline divides into right and left. Two horizontal lines on each side divide the area into three zones. First lies above the line of clavicle; second between line of clavicle and line at umbilical level; third below the level of umbilical line. First drains into head and neck lymph nodes; second drains into axillary nodes; third drains into inguinal/groin nodes. Malignancy drains to their respective nodes which depends on the location. Lesion on the line can spread to both territory lymph nodes. In skin and appendageal cancers, deep fascia also should be cleared (**Fig. 7.2**).

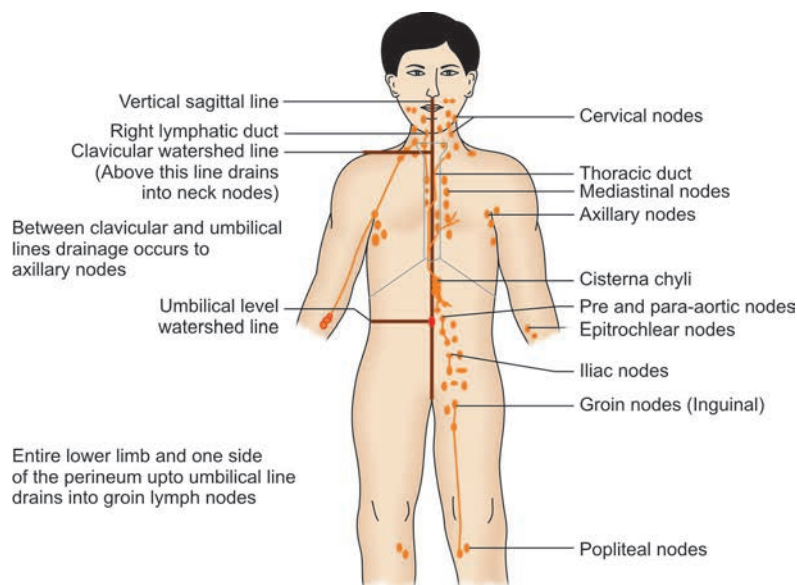


Fig. 7.2: Watershed zones/areas of lymphatic drainage. Vertical sagittal midline; clavicular horizontal line; umbilical line-are used to divide areas into three zones on each side. Above clavicular line drainage occurs to cervical nodes; between clavicular line and umbilical line drainage occurs into axillary nodes; below umbilical line drainage occurs to inguinal lymph nodes. At the line, drainage can occur to lymph nodes on either side.

Microanatomy of Lymph Node

Lymph node contains three regions—cortex; paracortex and medulla. Cortex contains mainly follicles. It may be rounded lymphocytic aggregations of primary follicles or lymphocytic aggregation with germinal centers of secondary follicles due to antigenic stimulation. It contains B lymphocytes, macrophages, dendritic reticulum cells. Germinal center is surrounded by small B lymphocytes. Both cortex and medulla are associated with humoral immunity. Proliferation of germinal centers suggests active humoral immunity with antibody production. Central medulla contains mainly lymphatic sinuses, arteries and veins, plasma cell and B lymphocytes. Paracortex is located in a zone between cortex and medulla. It contains T lymphocytes, related to cell mediated immunity. Post-capillary venules with high endothelial cells and lymphocytes in the wall are typical. In cell mediated immunity, paracortex expansion occurs. Afferent lymph vessels enter the node through the capsule. It enters the marginal sinus, communicates with intranodal sinus, merging as efferent lymph vessels which enter the hilum. Intranodal sinus lining is highly phagocytic containing littoral cells and sinus lining histiocytes. Main artery and veins pass through the hilum to enter the medulla, paracortex and inner part of cortex. Superficial cortex is supplied by direct capsular vessels.

Function

Most of the intravascular proteins are daily filtered through lymphatics and return to circulation again. Macromolecules and microbes are also filtered at the nodal level as first immune system. From GIT fat is absorbed directly through lymphatics. Lymph shows centripetal flow. Transport is mainly due to intrinsic contractility of the lymphatic vessels which contain valves for effective forward flow. To a lesser extent only other factors like muscle contraction, arterial pressure, thoracic pressure, respiratory movements play role.

History taking includes:

Name:

Address:

Age:

Sex:

Occupation:

Tuberculous lymphadenitis occurs in young age group. It is common in neck nodes. Hodgkin's lymphoma occurs in both young and elderly with bimodal age occurrence. Malignant secondaries in lymph nodes occur in old age. Non-specific adenitis, HIV infected lymphadenopathy can occur in any age group. Filarial lymphadenitis is common in any age group especially in certain parts of India and other developing countries. It commonly affects inguinal lymph nodes. Primary lymphoedema occurs in younger age group; secondary lymphoedema occurs in middle aged and elderly (**Figs 7.3 to 7.10**).

Filarial lymphoedema is more common in tropical countries. In India it is common in coastal areas. Orissa is the most affected state in India.



Fig. 7.3: Bilateral axillary lymph nodes—lymphoma. Entire lymphatic system has to be examined thoroughly.



Fig. 7.4: Tuberculous cold abscess neck.



Fig. 7.5: Fungating secondaries in the neck.

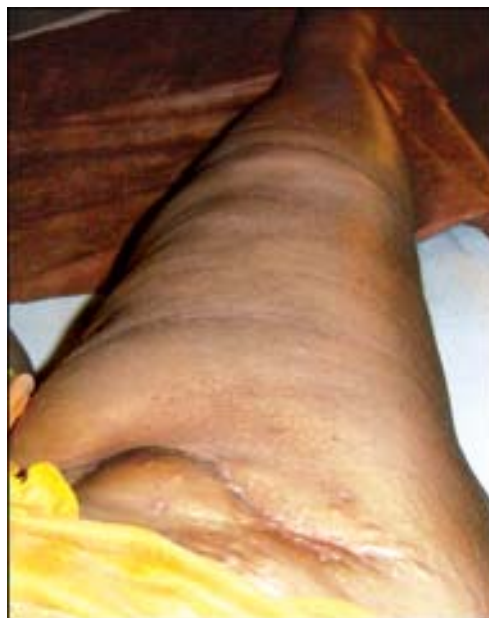


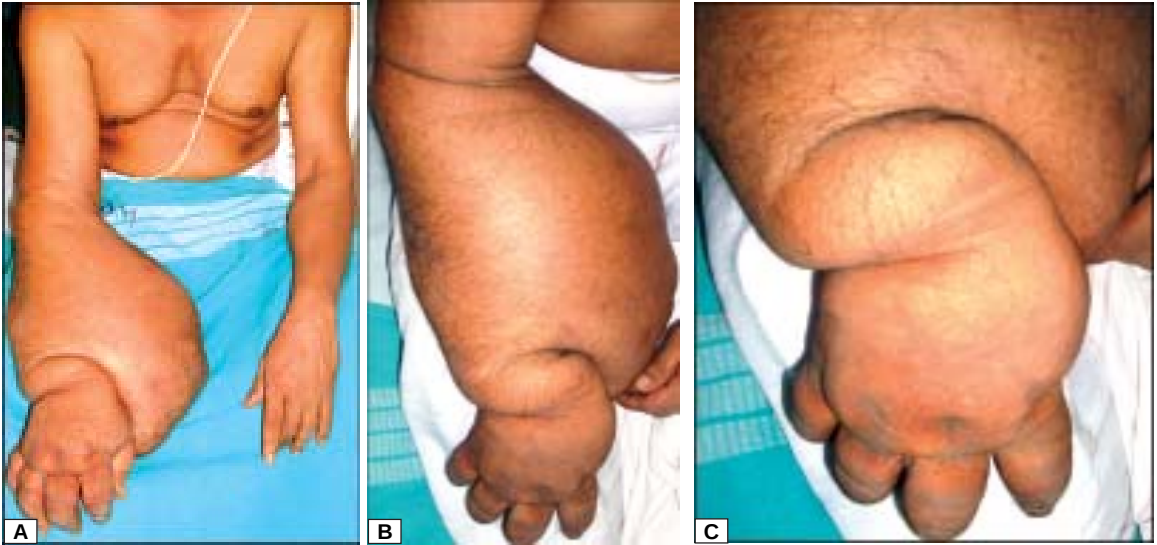
Fig. 7.7: Lymphoedema left lower limb after left sided ilioinguinal block dissection.



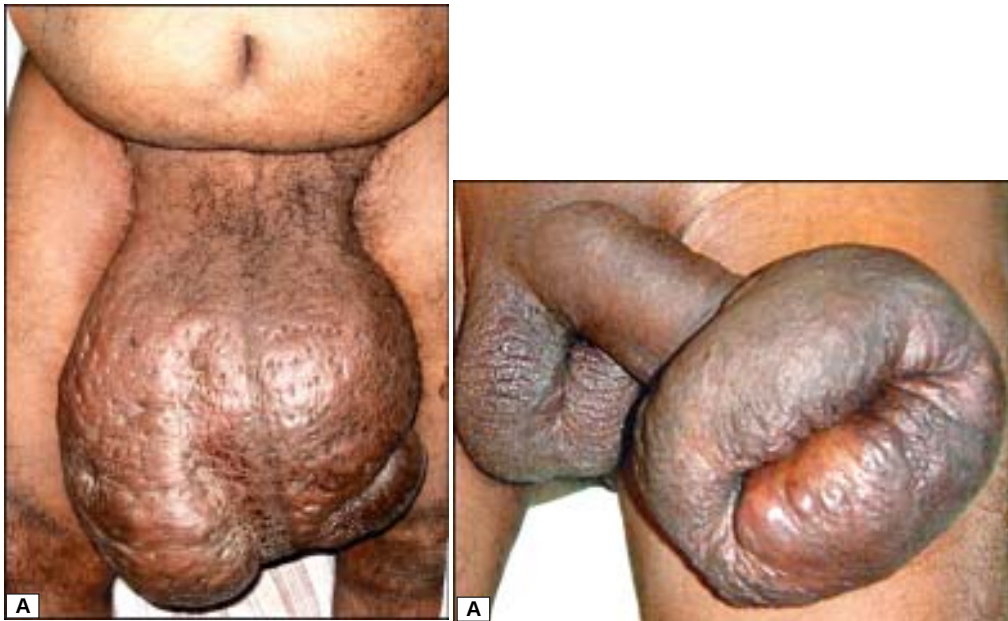
Fig. 7.6: Filarial lymphoedema—elephantiasis left leg. Also upper limb lymphoedema left side.



Figs 7.8A and B: Lymphoedema left sided in a male patient due to studded advanced cancer in left axillary nodes and chest wall. In photo B, female patient having lymphoedema right upper limb following mastectomy with axillary clearance.



Figs 7.9A to C: Right upper limb lymphoedema in a male who underwent reduction surgery for the same.



Figs 7.10A and B: Lymphoedema of scrotum and penis in two different patients.

Lymphoedema, primary or secondary is more common in females.

Chief Complaints

History of swelling and duration.

History of pain and duration.

History

History of Present Illness

Swelling

Swelling is the common presentation in lymphadenopathy. *Which group enlarged* first has to be noted. Its progress, presence of pain, whether reduced in size has to be noted. Enlarged lymph node if reduced in size after some time means it is of inflammatory origin; there is no spontaneous reduction in size in neoplastic conditions. *Number* of swellings is also important. Lymphoma may show multiple groups of nodal enlargement. *Site* of the origin of first swelling is important. If it is in the upper neck, then probably it may be either due to tuberculosis or primary in the oral cavity/pharynx/larynx. If it is in the lower neck then primary may be in oesophagus/bronchus, etc. Acute lymphadenitis is of short *duration* with pain and fever (with features of acute inflammation—redness, warmth, pain, and loss of function at the site). Malignancy in the lymph node either primary lymphoma or secondaries (metastases) are also of short duration (in few weeks). But initially it is painless. It is rapidly progressive and later may become painful. Tuberculosis, syphilis, brucellosis and sarcoidosis are of long duration and commonly painless. In syphilis lymph node enlargement occurs in secondary syphilis. Syphilis, brucellosis, sarcoidosis are rare now. Tuberculous lymphadenitis is still common condition in developing countries like India. Tuberculous lymphadenitis is much more common in HIV infected or immunosuppressed patients.

Pain

Acute lymphadenitis is painful. Tuberculosis is painless. If there is secondary infection pain can occur. Malignant lymphoma and secondaries in lymph node are initially painless but can be painful once there is fixity, necrosis, infiltration into deeper planes and nerves or fungation or secondary infection.

Fever

Continuous high grade fever occurs in acute lymphadenitis with suppuration. Evening rise of temperature is seen in tuberculous disease. But many patients with tuberculous lymphadenitis may not show any fever (fever is not always seen in tuberculous lymphadenitis). In Hodgkin's lymphoma intermittent fever (Pel-Ebstein fever) may be present which also decides prognosis and staging. Pel-Ebstein fever is also seen in brucellosis. Fever is not common in secondaries but can occur due to sepsis, tumour necrosis, and fungation.

Other Relevant History

History of cough, haemoptysis, chest pain in tuberculosis (tuberculous lymphadenitis may often be associated with pulmonary tuberculosis); *hoarseness of voice* due to pressure on recurrent laryngeal nerve either by lymph nodes in tracheo-oesophageal groove or by mediastinal nodes on left side compressing the left recurrent laryngeal nerve; *dysphagia* due to compression over oesophagus; swelling of face and neck due to compression of superior vena cava by superior mediastinal lymph nodes; *stridor or dyspnoea* by pressure on the trachea or bronchus. If nodes are secondaries, hoarseness/dyspnoea/dysphagia are may be features of primary tumour in larynx/bronchus/pharynx or oesophagus. Often tuberculous cervical lymphadenitis may be associated with laryngeal tuberculosis causing hoarseness of voice. Upper limb oedema may be present in axillary node enlargement due to compression. Inguinal lymph node enlargement may cause lower limb lymphoedema or venous oedema due to compression or lymphatic block or infiltration by malignant lymph node. Retroperitoneal nodal enlargement can cause compression/encasement of IVC/iliac vessels causing oedema of lower limbs.

History of bleeding gums is common in leukaemia, lymphomas, and blood dyscrasias.

History of loss of appetite and reduced weight is important in lymphomas, advanced secondaries, AIDS, etc.

History of trauma is often important in acute lymphadenitis.

History of night sweats, rigors, pruritis, and bone pain are important symptoms in lymphoma. Bone pain may be observed in the sternum, ribs, vertebra, etc. Jaundice may suggest liver secondaries; haemoptysis and chest pain may suggest lung secondaries; bone pain could be due to metastases.

Past History

Past history of any disease like tuberculosis, treatment received, earlier investigations like chest X-ray, FNAC, biopsy. Earlier treatment for malignancy with radiotherapy or chemotherapy should be asked for.

Personal History

History of smoking—its duration, number of cigarettes; alcohol intake—duration, quantity; chewing pan—duration, placing quid in the cheek; snuff abuse; dietary habits; history of sexual contact in case of HIV infection, syphilis, etc.

Family History

History suggestive of any disease or treatment taken for any specific condition by the family members is important. Tuberculosis can occur among many family members. Lymphoma can run in families.

General Examination

Detailed general examination is very essential. Anaemia/oedema/jaundice/clubbing/lymphadenopathy should be noted. Radial pulse/blood pressure/raise in temperature must be recorded. Attitude of the patient/nutritional assessment by skin texture, subcutaneous fat, weight, body mass index/any other relevant findings should be mentioned. Cachexia signifies advanced malignancy or tuberculosis. Increased pulse rate and fever suggests swelling with inflammatory pathology.

Local Examination

Inspection

Swelling

Detailed inspection for swelling which is discussed in swelling chapter should be done. *Number*—multiple lymph nodal enlargement occurs in lymphoma, tuber-

culosis, lymphatic leukaemia, sarcoidosis, brucellosis etc; *Size*—It is important in staging metastatic nodal status (N stage); *Shape*—Globular, hemispherical, oval; *extent* from a bony part, *Surface*—Smooth in lymphoma and tuberculosis, irregular in secondaries; *Margin*—Well-defined or ill-defined; *Pulsation*—As transmitted pulsation due to compression over adjacent major arteries like aorta/femoral/abdominal aorta; *Peristalsis*—May be visible in mesenteric lymphadenitis causing subacute obstruction; *Impulse on coughing*—may be present in swellings in relation to cavities like thorax; *Dilated veins over the swelling*—May be visible due to compression over the major veins in the neck or SVC in the mediastinum; *oedema* over the swelling or distal to it like in the limbs may be seen; *venous engorgement* of face can be seen in neck swelling (**Fig. 7.11**). Tuberculosis and Hodgkin's lymphoma usually occurs in the neck lymph nodes; filariasis and lymphogranuloma venereum (LGV L1, 2, 3) occurs in groin lymph nodes. Epitrochlear and suboccipital nodes are involved in secondary syphilis. Epitrochlear nodes also can get involved in Non-Hodgkin's lymphoma.



Fig. 7.11: Dilated veins in the neck left sided with enlarged lymph nodes. It could be lymphoma/secondaries with mediastinal node enlargement compressing SVC.

Skin over the swelling is red, inflamed, oedematous in acute lymphadenitis. It may be tense, shiny with often dilated veins in lymphoma. Skin ulceration, skin adherent to swelling underneath, fungation is common in secondaries in lymph nodes. Scar, sinus, ulcer may suggest tuberculosis or malignancy.

Features suggestive of pressure effects should be looked for:

Diffuse swelling of face and neck with dilated veins in this region suggests compression over major veins in neck or SVC obstruction in mediastinum. Axillary lymph nodes when enlarged may cause upper limb venous oedema. Neck nodes causing compression over subclavian vein also cause similar effect. Compression on hypoglossal nerve (causes deviation of tongue towards the same side with hemiparesis of tongue muscles of same side)/spinal accessory nerve palsy (causes defective shrugging of shoulder against resistance with wasting of trapezius muscle)/cervical sympathetic nerve (causes Horner's syndrome—enophthalmos due to decreased aqueous humour and pressure, miosis, anhidrosis, ptosis and loss of ciliospinal reflex) can be evident in large fixed neck nodes due to secondaries. Tracheal compression by the neck nodes causes stridor. Lower limb oedema may be evident in iliac or caval nodal enlargement (Fig. 7.12).



Fig. 7.12: Related neurological involvement (nerve infiltration by malignant tumour) causing altered sensory and motor functions. Example hypoglossal nerve involvement by neck secondaries can cause deviation of tongue towards same side.

Palpation

Normal lymph nodes are usually not palpable.

Local Rise of Temperature

It is seen in acute lymphadenitis, and often in vascular tumours like lymphoma. Lymph node secondaries may show local rise in temperature when once infection develops in it. Increase in vascularity also may add for the cause (Fig. 7.13).

1. **Tenderness** over the swelling is present in acute lymphadenitis, advanced/late stage secondaries. Enlarged nodes due to tuberculosis, syphilis and sarcoidosis are usually nontender.
2. **Number, size, shape** and extent should be assessed by palpation. Symmetrical and consecutive group of lymph node involvement is seen in Hodgkin's lymphoma. Asymmetrical involvement of lymph nodes is common in NHL. It is better to measure the swelling in two dimensions. Margin is assessed whether it is well-defined or ill-defined. Often some part of the margin may be clear and in such occasion the margin which is not clear should be mentioned. In the neck if lower margin is not clear then it is considered that it may be extending into the superior mediastinum.
4. **Surface** should be felt whether it is smooth or nodular. It may be smooth in lymphoma, nodular

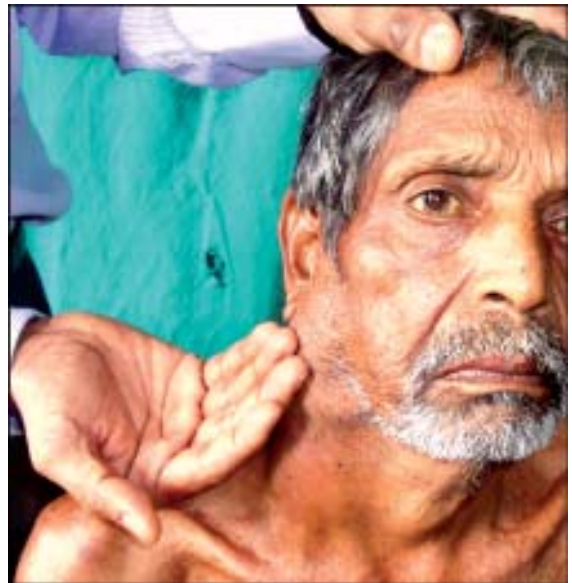


Fig. 7.13: Warmness should be looked for in lymph node enlargement.

in secondaries, typically matted in tuberculous lymphadenitis (due to periadenitis in caseating tuberculous lymphadenitis). Lymph nodes may be adherent to each other in lymphoma and secondaries. Discrete lymph nodes are often observed in lymphoma and hyperplastic tuberculous lymphadenitis. Discrete lymph nodes are also observed in lymphatic leukaemia, sarcoidosis, brucellosis, HIV infection and syphilis.

4. **Consistency** is very important finding to decide the pathology of the lymph node. It may be soft and fluctuant in cold abscess, suppurated lymph node or where there is tumour necrosis usually over summit (here remaining part of the swelling may be hard). In Hodgkin's lymphoma it has typical *India rubber consistency* with firm and elastic nature. In Non-Hodgkin's lymphoma it may be soft/firm/hard or variable in consistency. Shotty discrete lymph nodes are observed in syphilis.
5. **Mobility** of swelling should be checked in two perpendicular directions. Once it is checked muscle in relation to it should be contracted and mobility should be checked to find out the fixity.
6. **Fixity** of swelling can occur to overlying skin; adjacent muscle either superficial or deep; deep fascia; bone in the deeper plane; vessels and nerves. Swelling is non-mobile when nodes are fixed to bone in deeper plane. Swelling when adherent to the muscle show mobility, but mobility reduces when the muscle is contracted. Skin fixity is checked by moving the skin over the swelling or by pinching the skin (**Fig. 7.14**).
7. **Fluctuation test** is important when it is soft or tensely cystic. Fluctuation is observed in cold abscess (tuberculosis), when there is suppuration in lymph node with abscess formation, and when there is tumour necrosis. It is done with fingers after fixing the swelling or by Paget's method of eliciting the fluctuation (**Fig. 7.15**).
8. **Plane of the swelling** should be assessed. Whether it lies superficial/deep to deep fascia; deep to muscle should be assessed. It is done by stretching the deep fascia or by contracting the muscle underneath against resistance.
9. **Involvement of neurovascular bundle** should be assessed. Carotid and superficial temporal artery



Fig. 7.14: Skin over the swelling is adherent to node underneath or not should be checked by pinching the skin.



Fig. 7.15: First mobility of the node/swelling is checked in relaxed position. Muscle underneath is then contracted against resistance to check the change in mobility. If node is adherent to muscle, mobility will be restricted.

pulsation in neck nodal mass; femoral artery or distal arteries of the lower limb in groin nodes; radial artery pulsation in axillary nodes should be checked. Infiltration to adjacent nerve should be assessed by checking altered sensory and motor function (**Figs 7.16 and 7.17**).



Fig. 7.16: Palpation of carotid pulse. Neurovascular bundle should be examined in nodal enlargement.



Fig. 7.17: Pulse distal to the node should be examined for compression at the nodal level. Palpation of superficial temporal artery is done in neck node enlargement.

10. **Transillumination test** is negative in most of the lymph nodal enlargement. Only cystic hygroma in infants and acquired lymph cyst in any age group are brilliantly transilluminant.
11. **Transmitted pulsation** may be evident in large node sitting on the major artery. It is confirmed

by placing two fingers on the swelling. In transmitted pulsation fingers move only perpendicular (as raised without separating apart and away) over the surface but not apart. In expansile pulsation (due to arterial disease like aneurysm) fingers deviate apart (raised and separated) properly. Para-aortic nodes show transmitted pulsation due to close proximity to the aorta. The pulsation is checked in supine position and later confirmed in lateral and knee elbow position. In transmitted pulsation (nodal mass), pulsation reduces or disappears in changed positions whereas expansile aortic pulsation will remain same as before.

Drainage Area

Drainage area of lymph nodes should be examined. It suggests the origin of the disease in the lymph node (secondaries from carcinoma or melanoma/tuberculosis/lymphadenitis) from a primary focus in the drainage area.

Lymph Nodes in the Groin

They are divided into superficial and deep. Deep is called as *Cloquet's node*. It drains from glans penis or clitoris. Superficial lymph nodes are divided into vertical and horizontal groups. Vertical drains from lower limb. Horizontal chains are divided into medial and lateral. In carcinoma penis inguinal nodes are divided into five zones (**zones of Rouviere**) by a vertical and horizontal line centering at saphenous opening. Zone 1-superolateral; zone 2-superomedial; zone 3-inferomedial; zone 4-inferolateral; zone 5 is central. Superomedial zone 2 contains sentinel *sapheno-epigastric node of Cabanas*. They are palpated in supine position in relation to inguinal ligament in relaxed position (**Fig. 7.18**). Inguinal lymph nodes drain (from umbilicus to toes) entire lower limb, perineum, penis, scrotum, vulva, anus, buttock, lower anal canal, lower urethra, vagina, skin over lower abdomen below the umbilicus.

Axillary Lymph Nodes

They drain entire upper limb, trunks, breast, and chest wall from the clavicle to umbilicus. Axillary lymph nodes are divided into three levels in relation to



Fig. 7.18: Inguinal lymph node palpation.

pectoralis minor muscle. **Berg's levels**—Level I: Below the pectoralis minor; Level II: behind the pectoralis minor; Level III: above the pectoralis minor.

Examination of Axillary Lymph Nodes

Patient will be in sitting position in a stool. Right axilla is palpated using left hand and vice versa. Both axillae should be examined always (**Fig. 7.19**). Conditions



Fig. 7.19: Axilla should be inspected with arm raised above in sitting position.

which cause axillary lymph node enlargement are carcinoma breast; tuberculosis; lymphoma; lymphadenitis; any inflammatory or neoplastic pathology in upper limb, trunk, above umbilicus. While palpating number, size, surface, discrete or adherent, tenderness, consistency, mobility, fixity should be assessed. For staging in carcinoma breast (N staging), fixity/mobility of nodes, whether discrete or not are important features to be assessed. Which groups are enlarged is not important for staging. Palpable nodes are commonly significant in carcinoma breast; but non-palpable situation does not confirm the absence of metastases. 50% of clinically impalpable axillary nodes show histologically positive features after axillary dissection in carcinoma breast (**Fig. 7.20**).



Fig. 7.20: Carcinoma breast with secondaries in axillary nodes.

Palpation of Axillary Lymph Nodes

Anterior group (pectoral group) is situated behind the anterior axillary fold. The patient's arm is raised from her/his side and extended fingers of the right hand (for left axillary node) is passed into the axilla and insinuated beneath the pectoralis major. Pulp of the fingers is directed forwards and arm of the patient is lowered to rest relaxed over the forearm of the examiner's right hand. Pectoral nodes are palpated between thumb in front and fingers behind the muscle (**Figs 7.21A and B**).



Figs 7.21A and B: Examination of pectoral nodes.



Figs 7.22A and B: Examination of medial/central group of lymph nodes.

Central group (medial group) is over the lateral thoracic wall. The patient's arm is raised from side and extended fingers of the right hand of the examiner are passed high up to the apex of left axilla of the patient. Palm and fingers are directed towards the lateral thoracic wall. Patient's arm is relaxed down and forearm rests and relaxed on the examiner's forearm. Non-examining hand (left hand) of the examiner is placed on the right shoulder of the patient to steady and control the examination. Hand and fingers in the axilla are still pushed high up; hand is cupped with fingers sliding and moving over the lateral thoracic

wall to feel the slipping of the lymph nodes between fingers (**Figs 7.22A and B**).

Lateral/brachial axillary nodes are situated over the axillary vein. After placing the hand and fingers high in axilla, palm and fingers are directed laterally over the humerus beneath the insertion of the pectoralis major over third part of axillary vessels. Opposite hand of the examiner depresses the patient's shoulder for better assess.

Subscapular lymph nodes are located in the posterior axillary fold in relation to latissimus dorsi muscle. It



Fig. 7.23: Examination of subscapular group of lymph nodes.

is examined from behind. Examiner stands behind the patient. Respective examiner's hand is used for palpation of respective side of the subscapular group of lymph node. Hand and fingers are placed over the anteroinferior aspect of the posterior axillary fold and using other hand patient's arm is partially lifted. Nodes are palpated between thumb and fingers (**Fig. 7.23**).

Apical group of lymph nodes are palpated using examiner's opposite hand. Fingers are pushed very high up and another hand of the examiner is placed over the same shoulder of the patient to depress downwards.

Cervical Lymph Nodes

Cervical lymph nodes drain from lymphatics of head, neck, face, oral cavity, nasal cavity, paranasal sinuses, pharynx, larynx and thyroid. Left supraclavicular nodes receive from left upper limb, left side chest wall, left breast, abdomen and both testes. Cervical lymph nodes can be superficial or deep. Nodes are placed in different levels—Level I to level VI. Level VII is mediastinal node. Level I—submental and submandibular nodes; level II is upper deep cervical; level III is middle deep cervical; level IV is lower deep cervical; level V is posterior triangle nodes; level VI is central nodes (paratracheal and laryngeal). Level I and level II are further divided into a and b. Ia is submental; Ib is submandibular. Level Va above the spinal accessory level; level Vb is below it (**Figs 7.25 to 7.27**).



Figs 7.24A and B: Inner Waldeyer's ring should be examined in lymphadenopathy. It is significant in tuberculosis and NHL.

Inner Waldeyer's ring—adenoids, tubal tonsils, faucial tonsils, lingual tonsils also should be examined (**Figs 7.24A and B**).

Outer Waldeyer's ring—retropharyngeal lymph nodes; jugulodigastric lymph nodes; submandibular lymph nodes; submental nodes.



Figs 7.25A to D: Palpation of submental and submandibular lymph nodes on both sides.

In generalised lymphadenopathy, all nodal groups on both sides should be examined. Epitrochlear and popliteal nodes should be examined. These nodes may get enlarged in NHL. Epitrochlear node also may be enlarged in syphilis. Epitrochlear nodes are examined in sitting position with elbow partially flexed; 2 cm above the medial epicondyle in the groove between biceps and brachialis (**Figs 7.28A and B**). Popliteal

nodes are palpated ideally in prone position with knee flexed to relax the popliteal fascia. It is felt in the lower part of the popliteal fossa over the upper part of flat tibial surface. Lungs should be examined for pleural effusion. Para-aortic nodes, iliac nodes, liver and spleen enlargement should be looked for (**Figs 7.29A and B**). Examination of spine is also mandatory.



Figs 7.26A to E: Examination of level II, III, IV, V and supraclavicular nodes in the neck.



Fig. 7.27: Opposite side neck also should be always examined for any enlargement of lymph nodes.



Figs 7.28A and B: Epitrochlear lymph node palpation—2 cm above the medial epicondyle.



Figs 7.29A and B: Iliac and para-aortic nodes should be examined in generalised lymphadenopathy. Iliac nodes are palpated above and medial to inguinal ligament. It is enlarged in lymphoma, secondaries, etc. Para-aortic nodes are palpated in epigastrium above the umbilicus. It is resonant, non-mobile mass, vertically placed. It is felt on deep palpation.

Percussion

Sternal tenderness should be checked by direct method. It is elicited in lymphoma and leukaemias. Change in percussion note over the sternum (direct or indirect method) suggests superior mediastinal lymph node mass or other superior mediastinal tumors like retro-sternal goitre, thymoma, and aneurysm. Percussion over the abdomen to look for free fluid is essential. Percussion in respiratory system is done to find out pleural effusion (**Figs 7.30A and B**).



Figs 7.30A and B: Sternum should be percussed for tenderness and note. Sternal tenderness is significant in lymphoma and leukaemia. Normal note on percussion over the sternum is resonant. It becomes dull if there is mass lesion like lymph nodes in the superior mediastinum. Percussion is done by direct method to check tenderness. To find out the note either direct or indirect method may be used.

Auscultation

Auscultation over the mass is done to find out any bruit due to compression.

Systemic Examination

Respiratory system examination is done to look for pleural effusion, or any altered breath sounds; abdominal examination is done to look for palpable

liver, palpable spleen, para-aortic nodes, iliac nodes, etc. (**Figs 7.31A to E**).

Spine is examined for tenderness, paraspinal spasm, restricted spine movements and neurological deficits. NHL can involve spine causing neurological deficits (**Figs 7.32A and B**). There may be altered sensation, altered muscle power in the lower limb with urinary incontinence. It needs urgent radiotherapy/steroid therapy/surgical decompression.

Investigations

Blood

In acute lymphadenitis leucocytosis with neutrophilia is observed. Lymphocytosis is common in tuberculosis, lymphomas, leukaemia. Peripheral smear may show atypical lymphocytes in lymphatic leukaemia. Nocturnal blood smear may show microfilaria in peripheral smear of patient with filariasis. Specific blood tests for lymphogranuloma venereum or syphilis or HIV may be carried out. ESR is raised in tuberculosis and malignancies. Liver function test is useful in lymphoma to predict the possible involvement of liver. It is also useful during treatment period in case of tuberculosis to assess side effects. Hb% is significant in tuberculosis, lymphoma and secondaries. Platelet count is needed prior to therapy in case of lymphoma.

FNAC/Aspiration

It is useful in tuberculosis, malignancy. Caseating material with epithelioid cells is typical feature of tuberculosis. Langhans giant cells, lymphocytes and plasma cells are also found. Secondaries are diagnosed by FNAC. FNAC is not much useful in lymphomas as open biopsy is better to assess the type and to do histochemistry.

Lymph Node Biopsy

It is very useful method of investigation in lymph node enlargement especially in lymphomas. It is also useful in tuberculosis. In metastatic lymph nodal disease, if repeat FNAC is still not conclusive then only open biopsy is done. Routine open biopsy of secondaries in lymph node is avoided as spread can occur to further level of nodes increasing the nodal staging of the disease. Biopsy is done under general anaesthesia.



Figs 7.31A to E: Respiratory system is examined for effusion and altered breath sounds. Abdomen should be examined for liver enlargement, palpable spleen, para-aortic nodes.



Figs 7.32A and B: Spine should be examined in generalised lymphadenopathy for tenderness, paraspinal spasm. It could be lymphoma, chronic lymphatic leukaemia, spine tuberculosis.

Proper selection of lymph node to be taken for biopsy is essential so that possibility of negative result and need for rebiopsy may be reduced. Large sized/hard lymph node is more likely to be positive than small and soft lymph node. Adequate incision, exposure, retraction of deep fascia and soft tissues are needed. Breaking of the capsule is avoided as much as possible. Unnecessary handling of the node during dissection is avoided. Lymph node is held with nontoothed dissecting forceps. Ideally entire one lymph node is removed for biopsy. But in adherent node it is often difficult to remove the entire lymph node. Imprint films may be taken for cytological study. In tuberculosis cut section of node is yellowish, opaque with caseation in the centre. Histologically it shows caseating necrosis, epithelioid cells (modified histiocytes), *Langhans giant cells*, fibrosis, and chronic inflammatory cells. Cut section in lymphoma is fleshy, firm, elastic, grayish with often areas of haemorrhage and necrosis. Histologically (in HL) it shows cellular pleomorphism with features of anaplasia, lymphocytes, lymphoblasts, large multinucleated *Reed-Sternberg giant cells* with owl eye nuclei. Stroma shows silver stained reticular elements.

Radiological Examinations

Chest X-ray

Chest X-ray is significant to see pulmonary tuberculosis, pleural effusion, mediastinal lymph nodal

mass, calcified tubercular lymph node, primary bronchogenic carcinoma.

Relevant investigations for primary like endoscopies, blind biopsies, CT of the part is also done.

CT Chest

CT chest is more relevant than chest X-ray to detect early lesions either malignancy or inflammatory condition and also mediastinal nodes. 30% of lesions in the lungs can be missed in chest X-ray but are well detected by CT chest. CT abdomen is done when needed in individual patient basis.

US Abdomen

US abdomen is done to see liver, spleen and para-aortic, mesenteric, iliac nodes especially in lymphomas. *Dancing filaria* may be evident if US is done directly on the lymph node. US of specific area like axilla/groin/neck is done to assess the size, extent, relations of enlarged lymph nodes and also vascularity, and relation of major vessels in the region.

Lymphangiography

It is done in congenital lymphoedema to see aplasia/hypoplasia/hyperplasia; lymphomas (shows reticular pattern) to assess the response for treatment as dye stays for long duration in the lymph node and so node can be assessed by taking repeated X-rays of the area. Patent blue dye or 1 ml isosulphan blue is

injected subcutaneously in the web space of the foot. Lymph vessels take up this dye and make it clearly visible. Using operating microscope, after skin incision, lymphatic vessel is identified and cannulated with 30 G needle. Ultrafluid (ethiodised oil) lipiodol is injected slowly using pressure pump at a rate of 1 ml in 8 minutes. Total of 7 ml of contrast agent is injected. It takes 24 hours to pass through the lymphatics and reach the lymph nodes—iliac and para-aortic nodes. X-rays are taken to visualise lymphatics and lymph nodes. Lymphomas show foamy or reticular pattern. Secondaries show irregular filling defects. Lymphatic pattern/anomalies can be assessed properly and classify lymphoedema as congenital hyperplasia (10%); distal obliteration (80%); proximal obliteration (10%)—*Browse's lymphangiographic classification of lymphoedema*. Disadvantages—Procedure is invasive, technically difficult, time consuming, dye may not reach the required area, extravasation of dye can cause complications like sepsis, skin necrosis.

In melanoma radiopaque phosphorus is added to the dye during lymphangiography which will destroy malignant cells in lymph nodes and is called as *endolymphatic therapy*.

Isotope Lymphoscintigraphy

It has got 90% sensitivity; 100% specificity. It is useful to differentiate lymphoedema from other causes of limb swelling. It is simple, safe, and reproducible and there is low exposure to radioactivity (5 mCi). Radio-labeled human albumin or Technetium 99m labeled sulphur colloid is injected into the web space. It migrates in skin and subcutaneous lymphatics and is monitored using whole body gamma camera. It gives clear images of lymphatics, and nodes in the inguinal, iliac, para-aortic region. Later it gives image of thoracic duct also. Amount of radiotracer is assessed in the inguinal nodes in 30 and 60 minutes. Normal uptake is 0.6 to 1.6%. An uptake less than 0.3% in 30 minutes is diagnostic of lymphoedema. In oedema due to venous diseases, uptake is rapid and shows more than 2% in 30 minutes in inguinal nodes. Thoracic duct, liver and other lymphatic organs in the body can be visualised. It is technically easier and faster.

Mediastinal Gallium 67 radioisotope scan can be done to find out whether mediastinal nodes are involved or not.

Laparoscopy/mediastinoscopy/thoracoscopy are useful in difficult cases.

Bone marrow aspiration is essential once lymphoma is confirmed to stage the disease and also eventually to see the therapeutic response. It is also important in lymphatic leukaemia.

CT/MRI spine to see spine involvement in case of lymphoma.

Other Tests

Mantoux test/guinea pig inoculation test for tuberculosis; **Gordon's biological test** for Hodgkin's lymphoma; **Frei's intradermal test** for lymphogranuloma venereum. In **Gordon's test** affected lymph node emulsion is injected into the cerebrum of the rabbit which initiates encephalitis in few days in case of Hodgkin's disease. In **Frie's test** pus is collected from an unruptured bubo. It is diluted using saline—1:10; sterilised with 60 degree temperature. 0.1 ml of such solution when injected intradermally will show a reddish papule at the site of injection in case of positive for lymphogranuloma inguinale (LGV- L1, 2, 3).

Pathology of Lymph Systems

Generalised Lymphadenopathy

Generalised lymphadenopathy means enlargement of more than one non-contiguous group of lymph nodes for a period of 3 months with each group showing at least one node more than 1.0 cm in size.

Causes for generalised lymphadenopathy are tuberculosis; lymphoma either Hodgkin's or Non-Hodgkin's; lymphatic leukaemia; HIV infection; autoimmune diseases as part of collagen disease; secondary syphilis (secondary and primary syphilis; not in tertiary syphilis); infectious mononucleosis; sarcoidosis; brucellosis; toxoplasmosis, etc. Presently tuberculosis, lymphoma, leukaemia are common causes. Other causes can be present but rare. In generalised lymphadenopathy, all groups of lymph nodes should be carefully examined in detail—neck nodes; axilla; groin nodes. Epitrochlear nodes, popliteal nodes should also be examined. Nodes on both sides should be examined. Respiratory system and chest should be examined for change in breath sounds, pleural effusion. Abdomen should be examined

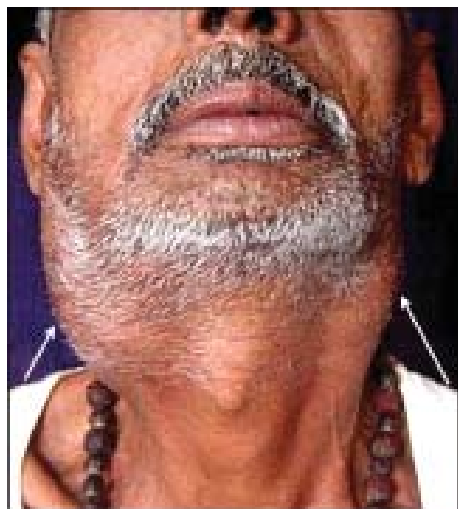


Fig. 7.33: Generalised lymphadenopathy in a patient with Non-Hodgkin's lymphoma.

for hepatomegaly, splenomegaly and ascites. Looking for sternal tenderness, percussion note on the sternum, and spine examination is must. Fever, itching, weight loss, wasting, jaundice, neurological deficits are important features to be noted (**Fig. 7.33**).

Causes of Lymph Node Enlargement

- a. Inflammatory:** *Acute lymphadenitis; Chronic lymphadenitis; Granulomatous lymphadenitis:* (a) *Bacterial* like tuberculosis, syphilis, tularaemia, brucellosis, lymphogranuloma venereum, Cat scratch fever. (b) *Viral* like HIV infection, infectious mononucleosis. (c) *Parasitic* like filarial adenitis, toxoplasmosis. (d) *Fungal* like blastomycosis, histoplasmosis, coccidioidomycosis. (e) *Other causes* like sarcoidosis.
- b. Neoplastic:** Lymphomas (HL and NHL); Secondaries in lymph node from most of the carcinomas, some sarcomas, malignant melanoma.
- c. Haematological:** Chronic lymphatic leukaemia.
- d. Immunological:** Serum sickness, drug reactions, rheumatoid arthritis, systemic lupus erythematosus, scleroderma, polyarteritis nodosa.

Acute Lymphadenitis

It is acute bacterial infection of the lymph nodes. It usually results following spread from an infective focus

in the drainage area like in neck nodes following tonsillitis, oral infection, scalp infection, nasal and ear infection; in the axillary nodes following skin infection, hidradenitis, trauma; in groin nodes following filarial adenitis with acute presentation, trauma (bare foot walk, ulcers, abrasions, wounds), infective focus in the lower limbs, perineal diseases. It is rapidly enlarging swelling of sudden onset (lymph node) with pain, fever, redness, tenderness, brawny oedema. Once suppuration (pus formation) occurs, fluctuation develops in the centre (Paget's test is positive). Pitting on pressure is appreciated in the periphery of swelling. Primary focus in the drainage area may be evident. If patient is immunosuppressed and diabetic septicaemia may often develop.

Chronic Lymphadenitis

It is usually due to non-specific bacterial infection. Lymph nodes are enlarged, discrete or often adherent, slightly tender and elastic. Focus may be from drainage area like scalp, limbs, and perineum. It should be differentiated from tuberculosis and lymphomas.

Reactive Hyperplasia of Lymph Nodes

It is enlargement of lymph node as hyperplastic response to existing diseases in the drainage area like carcinomas or recurrent infections. Lymph node is enlarged, non-tender, firm, discrete and mobile. Hyperplasia occurs in germinal centre of the node. But clinically it is difficult to differentiate it from secondaries from carcinoma. Histological study following FNAC/biopsy or after radical dissection confirms the reactive hyperplasia.

Tuberculous Lymphadenitis

It is common in *neck nodes*. Mediastinal, mesenteric, axillary and inguinal nodes also can get involved. In the neck, nodes are involved commonly through tonsils. *Upper deep cervical nodes* (54%) are commonly involved. Posterior triangle nodes are involved in 22% cases. Often multiple, bilateral nodes may get involved. Axillary nodes are often diseased through retrograde spread from neck nodes of posterior triangle or through blood or from apical lung disease across parietal pleura. Infection also may be following blood spread from primary pulmonary tuberculosis. Occasionally it may

be part of miliary tuberculosis also. It is caused by *Mycobacterium tuberculosis*. Infection is more common in HIV, lymphoma, malnourished, immunosuppressed patients. Bacteria evoke inflammation and cell mediated immunity in the paracortex. Disease passes through five stages. **Stage 1:** Stage of infection and lymphadenitis; **Stage 2:** Stage of periadenitis and matting; **Stage 3:** Stage of caseating necrosis and cold abscess formation; **Stage 4:** Stage of collar stud abscess formation where caseating material passes through deep fascia into subcutaneous tissue and gets adherent to the skin; **Stage 5:** Stage of sinus formation. Fibrosis and calcification can occur in this node. Tuberculous lymphadenitis can occur in two types.

Type 1: Caseating tuberculous lymphadenitis which is 80% common. It causes caseation, matting due to periadenitis, cold abscess and sinus formation. Here body resistance is less and drug may not reach in effective concentration into the area of caseation and so resistance and residual disease is common to develop. **Type 2:** Hyperplastic tuberculous lymphadenitis is 20% common. It is firm, nontender, discrete node without central caseation. Cold abscess and sinus will not occur. Host resistance is good and so shows good and rapid response to drugs. Gross features of caseating tuberculous lymphadenitis are firm, matted, node with yellowish central caseation on cut section. Histologically it contains *epithelioid cells* (are modified histiocytes—diagnostic feature), Langhans giant cells, macrophages and lymphocytes. Clinically, presents as firm swelling, which is not warm, nontender, matted, usually mobile, can be adherent to adjacent muscles. Cold abscess is soft, nontender, smooth, fluctuant, non-transilluminating, well localised, often nonmobile swelling with free non-adherent skin over the surface. Skin will be adherent at collar stud abscess stage. Tonsils and lungs should be examined for primary focus (**Fig. 7.34**). Secondaries, lymphoma, chronic lymphadenitis, lymph cyst, HIV, branchial cyst are differential diagnosis (**Fig. 7.35**).

Filarial Lymphadenitis

It is common in inguinal nodes. Firm, tender, enlarged lymph nodes are common. Periodic fever and pain is common. Thickening of spermatic cord (funiculitis), thickened epididymis (epididymitis), thickened scrotum, filarial limb are common. Night blood sample



Fig. 7.34: Tonsil is the common focus of the tuberculous lymphadenitis of cervical nodes.



Fig. 7.35: Tuberculous lymphadenitis with sinus in the neck.

may show microfilaria in the circulation. Eosinophilia is common. US of lymph node shows typical dancing microfilaria. Biopsy reveals adult worm.

Lymphogranuloma Inguinale/Climatic Bubol/Tropical Bubo

Lymphogranuloma inguinale/climatic bubo/tropical bubo is a sexually transmitted disease due to *lymphogranuloma inguinale*, a venereal spreading

organism (LGV, Chlamydia type L1, 2, 3). In LGV, lesion is small, painless and commonly unnoticed in *primary genital stage*. *Secondary stage* lesion develops in 2 weeks. In males inguinal lymph nodes; in females intrapelvic and pararectal nodes are involved. Eventually suppuration of inguinal nodes occurs leading into discharging sinuses. *Frei intradermal test* becomes positive in 6 weeks and remains positive for life time. *Tertiary stage*, eye, joints, meninges may get involved after many years. Repeated chronic inflammation, lymphatic blockage, scarring can cause rectal stricture and vulval elephantiasis (*esthiomene*) in females.

Soft Chancre/Soft Sore/Ducrey's Ulcer/Chancroid/Bubo

This is a *venereal* disease which presents with multiple irregular genital ulcers that appear 3 days after infection with bacteria, *Haemophilus ducreyi*. They are acute painful, tender, non-indurated ulcers. Floor shows yellowish slough with purulent discharge. Edge is oedematous and inflamed. Acute regional lymphadenitis with suppuration occurs presenting as tender, soft or firm swelling. Such soft fluctuant inguinal swelling is termed as *bubo*.

Infectious Mononucleosis (Glandular Fever)

It is an acute self limiting disease caused by *Epstein Barr virus* in young adults who presents with fever, sore throat, rashes, tender, elastic, lymphadenopathy, splenomegaly, abnormal lymphocytes in the peripheral smear, and subclinical hepatitis with altered liver function tests. Initially there is neutropenia, later leucocytosis develops. There are atypical mononuclear cells which are T cell reactive lymphoblasts. Lymphoma and lymphatic leukaemia are differential diagnoses. Blood picture may mimic lymphatic leukaemia. Heterotopic antibodies (*Paul-Bunnell antibodies*) develop in the serum which agglutinates sheep red cells. *Paul-Bunnell test* is diagnostic. Test becomes positive during early phase of infection and disappears in 2 months. So test is diagnostic of recurrent and new infections. Inactivated serum of patient is diluted to two times and mixed with equal quantity of 1% suspension of sheep erythrocytes to see agglutination of 100 or above.

Toxoplasmosis

It is caused by *Toxoplasma gondii*, intracellular protozoa, transmitted from mammals to humans who eat raw or underdone meat, causes generalised lymphadenopathy and fever in children. Congenital infection may cause life threatening neonatal jaundice, encephalomyelitis, hydrocephalus, microcephaly, blindness and intracerebral calcification.

Cat Scratch Fever

It is a type of *psittacosis* (psittacos means parrot) caused by *Chlamydia psittaci*. After cat scratch or droplet infection, it presents with inflammatory features at the site, fever, malaise, anorexia, regional lymph node enlargement after 2 weeks which suppurates with sterile pus inside that often bursts open. Skin test using human lymph node pus as antigen is diagnostic. It mimics chronic or tuberculous lymphadenitis. Pus, tissue smears, spleen, lungs, brain show LCL bodies. Flu like syndrome, fatal pneumonia, meningoencephalitis, endocarditis, pericarditis are dangerous complications carrying 20% mortality.

Syphilitic Adenitis

It occurs as generalised shotty, firm, discrete, non-tender lymphadenopathy especially epitrochlear and suboccipital lymph nodes. Often it is seen in primary syphilis also. It is not seen in tertiary syphilis. For detail please refer Chapter 2: Examination of an Ulcer.

Sarcoidosis

It is a differential diagnosis for lymph node mass. It is basically a *granulomatous* condition of unknown aetiology with bilateral hilar lymphadenopathy; along with involvement of lungs, liver, spleen, lymph nodes, lacrimal glands, parotid glands, CNS, associated with hypercalciuria, and acute onset of erythema nodosum in the skin. Fever and loss of weight are not common. It shows non-caseating granuloma with epithelioid cells, positive *Kveim-Siltzbach* skin test (80%); and high levels of serum angiotensin converting enzyme (SAGE). Investigations needed are CT chest; mediastinoscopy; nodal biopsy; slit-lamp examination of eye. Often shows abnormal immunoglobulins in the circulation. It is treated by corticosteroids with good

response. It should be differentiated from other causes of lymphadenopathy especially Hodgkin's lymphoma.

Secondaries in Lymph Nodes

Metastatic disease in regional lymph nodes occurs by lymphatic spread usually by permeation up to first nodal level and later by embolisation. Head and neck cancers account for 80% cases in neck nodes. In axilla, carcinoma breast is the common cause; others are skin malignancies in upper limb, chest wall, etc. Carcinoma in lower limb, perineum, penis, scrotum, genitalia spreads to groin lymph nodes. These lymph nodes are **stony hard (Fig. 7.36)**, with smooth surface, initially non-tender but soon become tender by tumour necrosis, nerve infiltration, and fungation (**Figs 7.36 to 7.38**). They are initially mobile, but eventually become fixed and nonmobile as it gets adherent to muscle, and bone. *Infiltration to regional major vessels* causes absence of pulsation (example carotid in neck), *nerve infiltration* causes neurological deficits (infiltration of hypoglossal nerve causes its palsy leading to deviation of tongue towards same side (**Fig. 7.37**) and wasting of tongue muscle of that side/spinal accessory nerve infiltration causes poor shrugging of the shoulder); *venous obstruction* causes oedema of distal part (groin secondaries can cause venous oedema of lower limb). Few sarcomas (rhabdomyosarcoma,



Fig. 7.36: Secondaries in cervical node. It is fixed advanced disease. It is stony hard in consistency.



Fig. 7.37: Advanced secondaries in neck with fungation and hypoglossal nerve palsy showing deviation of tongue towards the same side.



Fig. 7.38: Fungating secondaries in inguinal lymph nodes on the right side. It is an advanced disease.

synovial sarcoma) can cause secondaries in lymph nodes. Secondaries from malignant melanoma are usually pigmented.

Chronic Lymphatic Leukaemia

It is a haematological disorder with generalised lymphadenopathy, splenomegaly, bleeding tendencies (bleeding gums), fever, anaemia, decreased weight. Peripheral smear (lymphocytosis) and bone marrow aspiration is diagnostic.

Lymphomas

They are progressive neoplastic condition of lymphoproliferative system arising from stem cells. They are 3rd most common malignancy in children comprising



Figs 7.39A and B: Lymphoma in an old man and in a child. Both could be Hodgkin's lymphomas.

15% of paediatric cancers. It is often genetically pre-disposed. It is commonly associated with Sjogren's syndrome, Wiskott-Aldrich syndrome, ataxia telangiectasia, Epstein-Barr virus infection, celiac sprue, *H. pylori* infection (MALT lymphoma), ionising radiation (**Figs 7.39A and B**).

Types: Hodgkin's lymphoma (HL); Non-Hodgkin's lymphoma (NHL). **WHO modified REAL (Revised European American Lymphoma) classification of**

lymphoma—Type 1. B-cell neoplasms—*subtype I*—of precursor B cell—Acute lymphoblastic leukaemia (ALL), Lymphoblastic leukaemia (LBL); *subtype II*—of peripheral B cell—all B cell related NHL. **Type 2.** T cell putative NK cell neoplasms—*subtype I*—of precursor T cell—ALL, LBL T cell related; *subtype II*—of peripheral T cell and NK cell includes all T cell related NHL. **Type 3.** Hodgkin's lymphoma—*subtype I*—predominant HL-nodular lymphocyte type; *subtype II*—classical HL-nodular sclerosis, lymphocyte rich, mixed cellularity, lymphocyte depletion.

Hodgkin's Lymphoma (HL-Thomas Hodgkin)

It is the *commonest* type of lymphoma having fleshy, pinkish gray, rubbery lymph nodes on gross; with malignant lymphocytes, reticulum cells, histiocytes, giant cells with two large mirror image nuclei [*Reed-Sternberg giant cells* (RS cells are also observed occasionally in other conditions like glandular fever)] on microscopy. Predominant and classical HL are the types. **Rye's classification** includes lymphocytic predominance; mixed cellularity; nodular sclerosis (commonest); lymphocytic depletion. **Features:** It is common in males; common in young and elderly (bimodal); presents as painless enlargement of lymph nodes which are smooth, firm (*India rubber consistency*), non-tender. Neck is the commonest location (80%); commonly seen in lower deep cervical and posterior triangle nodes (**Fig. 7.40**). Axillary, media-



Fig. 7.40: Hodgkin's lymphoma in an adolescent boy.

stinal, inguinal, abdominal—are the other groups which may be involved. Consecutive and symmetrical involvement; *splenomegaly* (45%) is common. Hepatomegaly, jaundice, *constitutional symptoms* (stage B) like weight loss, fever, pruritus, anaemia, bone pain are other features. Mediastinal involvement may cause SVC obstruction. Bone involvement may present with sternal tenderness, vertebral pain. Anaemia, pancytopenia is common. **Ann Arbor clinical staging** (Ann Arbor is a place): *Stage I*: Confined to one group of lymph nodes; *Stage II*: More than one group of lymph nodes on one side of the diaphragm; *Stage III*: Nodes on both sides of the diaphragm; *Stage IV*: Extra nodal involvement like liver, bone marrow. 'S' is added to stage if spleen is involved; 'B' is added for presence and 'A' for absence of constitutional symptoms. 'E' is added for extranodal spread. Stage III (1) is nodes above the renal vein and stage III (2) is below. **Differential diagnoses** are tuberculous adenitis; NHL; HIV; chronic lymphatic leukaemia; non-specific adenitis; sarcoidosis; secondaries in lymph nodes. *Staging laparotomy* which was earlier done is not commonly advocated now. After opening abdomen, splenectomy is done to remove the tumour bulk and to avoid irradiation of splenic area which often causes pulmonary fibrosis. Biopsies are taken from both lobes of the liver (needle biopsy) and from para-aortic, celiac, mesenteric, iliac nodes. In females ovaries are fixed behind the uterus to prevent radiation oophoritis (ovarian translocation).

Non-Hodgkin's Lymphoma (NHL)

It occurs in middle-aged and elderly. It is more aggressive than HL. Lymph node involvement is asymmetrical and non-contiguous. General condition is poor. Inner Waldeyer, epitrochlear and peripheral nodes are commonly involved. Hepatomegaly is common. Spleen is not commonly involved. Vertebral involvement and paraplegia can develop which warrants radiotherapy for spine. Cachexia, secondary infection and immunosuppression are more common. *Rappaport and working classifications* are used. It can be nodular or diffuse. It can be B cell or T cell type. It can be precursor cell type or peripheral cell type. It can be small, large, cleaved, uncleaved, etc. It can be low grade, intermediate grade or high grade. Carcinoma or sarcomas can mimic NHL often (**Fig. 7.41**).



Fig. 7.41: Non-Hodgkin's lymphoma with fungation in the axilla.

Burkitt's lymphoma (Malignant lymphoma of Africa)

It is common in South Africa and New Guinea; common in children; *Epstein-Barr virus* may be the cause; often associated with infectious mononucleosis; common in malaria endemic area. It is common in jaw either upper or lower; neck nodes are commonly involved; multifocal, rapidly growing, painless lesion. Other group of lymph nodes also can be affected. Often bilateral renal involvement (75%) is common. Ovaries are commonly affected in females. Histology shows primitive lymphoid cells with large clear histiocytes (*starry sky pattern*). It can be *endemic African type*—common in jaw; *non endemic sporadic type*—common in abdomen; *aggressive type*—seen in HIV patients.

Cutaneous T Cell Lymphoma

Cutaneous T cell lymphoma comprises *mycosis fungoides*, *Sezary syndrome*, reticulum cell sarcoma of skin and other skin lymphocytic dysplasias. Mycosis fungoides is commonest among them. Cutaneous T cell lymphoma can be indolent (commonly mycosis fungoides); aggressive (*Sezary syndrome*); provisional (granulomatous/panniculitis like T cell lymphoma). Initial macular/patch/plaque phase slowly changes into tumour phase with painful, pruritic erythroderma often with visceral spread. Alopecia mucinosa and follicular mucinosis are common in mycosis fungoides. Lymph nodes may get involved. Tumour cells in peripheral smear are also important in deciding therapy and prognosis. Multiple skin biopsies/peripheral smear/node biopsy/immunohistochemistry/pheo or geno-

typing are important investigations. Prognosis depends on extent of skin involvement (more than 10% body surface area carries poor prognosis)/nodal spread/blood spread. *Sezary syndrome* is a type of cutaneous T cell lymphoma with skin lesions with special *Sezary cells* having cribriform nucleus. It is often associated with leukaemias. It is treated like any other cutaneous T cell lymphoma.

Lymphoedema

It is accumulation of fluid/lymph in extracellular and extravascular compartment, commonly in subcutaneous tissue. It is due to defective lymph drainage. It is accumulation of increased protein rich interstitial fluid.

Kinmonth classification

Primary: Less common; without any identifiable cause. It is common in females; common in lower limb and left side. It can be *Lymphoedema congenita*—present at birth, < 2 years (**Fig. 7.42**), familial type is called as *Nonne-Milroy's disease*; *Lymphoedema praecox*—present at puberty, between 2–35 years of age; 80% of primary Lymphoedema belong to this type; familial type is called as *Letssier-Meige's syndrome*; *Lymphoedema tarda*—Present in adult after 35 years of age, *Lymphangiographically* it can be hypoplasia (70%); aplasia (15%); hyperplasia/varicose lymphatics (15%).

Secondary: Most common; is due to a definitive acquired cause. *Causes* are—trauma; inguinal/axillary block dissections; filarial lymphoedema; tuberculosis, syphilis, fungal infection, advanced fixed nodal

malignancy in axilla or groin; radiotherapy; non-specific recurrent bacterial infection of nodes; rare causes like Rheumatoid arthritis, snake bite, insect bite, DVT, chronic venous insufficiency. *Wuchereria bancrofti* is the cause for filarial lymphoedema. Recurrent lymphangitis causes obliteration of lymph vessels → dermal lymphatic backflow → retrograde obliteration (or die back of lymphatics) → oedema initially pitting but later nonpitting → recurrent cellulitis—thickening of skin → accumulation of proteins, growth factor, glycosaminoglycons → activation of collagens and keratinocytes → protein rich lymphoedematous tissue formation → deposition of ground substance, subdermal fibrosis → dermal thickening and dermal proliferation → fissuring → cracks-ulceration-abscess formation → stout leg with unbearable weight → *elephantiasis*. Rarely it may cause protein losing diarrhoea, chylous ascites, chylothorax, chyluria, lymphorrhoea. Recurrent lymphadenitis occurs in the region which aggravates the condition. Disease in the limb is confined to skin and subcutaneous tissue, i.e. often, only superficial lymphatics are involved by the disease, deep lymphatics are not. Superficial and deep lymphatics are not communicating with each other (Unlike the veins in the limb where superficial and deep veins are freely communicating with each other). Sites of lymphoedema—(1) Lower limb—commonest. (2) Upper limb. (3) Scrotum and penis (*Ram's horn penis*). (4) Breast—requires reduction mammoplasty. (5) Labia. (6) Eyelid. (7) Localised lymphoedema. **Clinical features:** Swelling in the foot, extending progressively in the leg; *Buffalo hump* in the dorsum of the foot; squaring of toes; skin over the dorsum of foot cannot be pinched because of subcutaneous fibrosis—*Stemmer's sign* (**Figs 7.46A and B**); initially pitting oedema which later becomes nonpitting (**Figs 7.43A and B and 7.44**); eczema, fissuring, papillae formation, ulceration, lymph ooze, loss of normal perimaleolar shape → tree trunk pattern; elephantiasis (**Figs 7.45A and B**); recurrent fever; malaise; headache; athlete's foot; joint pain; limb disability; social discomfort are the features. **Brunner's grading of lymphoedema**—*Latent*—subclinical: No clinically apparent lymphoedema. *Grade I:* Pitting oedema which more or less disappears on elevation of the limb—is due to excess deposition of interstitial fluid. *Grade II:* Nonpitting



Fig. 7.42: Congenital lymphoedema right side in a young girl.



A



B

Figs 7.43A and B: Early lymphoedema left side. It is pitting in nature.

oedema develops which does not reduce on elevation. **Grade III:** Oedema with irreversible skin changes like fibrosis, papillae, fissuring. It may be **mild lymphoedema** - < 20% excess of limb volume; **moderate** - 20-40% excess of limb volume; **severe** - > 40% excess of limb volume.

Differential diagnoses: (1) Cardiac causes, hypo-proteinaemias, malnutrition, nephrotic syndrome. (2)



Fig. 7.44: Late lymphoedema—non-pitting in nature.

Myxoedema. (3) Trauma. (4) Venous diseases like DVT, etc. (5) Lipodystrophy and lipoidosis. (6) Arterial diseases including A-V malformations. (7) Gigantism. (8) Drug induced—steroids, oestrogens, nifedipine. **Complications:** Skin thickening; abscess formation; recurrent cellulitis; maggots' formation; nonhealing ulcers; septicaemia; lymphadenitis; lymphangiosarcoma (*Stewart-Treves syndrome* in post-mastectomy limb).

Acute Lymphangitis

It is the bacterial infection of lymphatic vessels from a focus in the draining area. Usually gram positive staphylococci and streptococci are the causative organisms. Clinically there will be fever, raised, thin, painful, tender, visible red streaks of lymphatic vessels which are obvious. Blanching on pressure is typical. Tender palpable regional lymph node in axilla or groin is common. It is common in upper and lower limbs. Primary focus may be small in digits/interdigital space/plantar aspect of foot/in the nails. Condition may lead into cellulitis once tissue planes get infected. Toxaemia, septicaemia may occur.

Chylous Ascites

It is collection of lymph in the peritoneal cavity. It is due to obstruction of intestinal lymphatics and leak.



Figs 7.45A and B: Elephantiasis leg in a young male and middle aged female.



Figs 7.46A and B: Changes in foot in lymphoedema—Buffalo hump; squaring of toes; non-pinachable skin over the dorsum (Stemmer's sign).

Causes: Congenital lymphatic abnormalities (in children commonest); malignancy either nodal secondaries or nodal primary causing obstruction (commonest cause in adult); tuberculosis causing block in lymph drainage and rupture and leak into the peritoneal cavity; filarial lymphoedema causing obstruction; post-surgical cause. **Features:** Ascites often massive; severe malnutrition and protein deficiency; features specific to the cause. Ascitic fluid aspiration is chalky white in colour and it shows chylomicrons. Triglyceride level more than 110 mg/dl is diagnostic. It should be studied for fat globules, proteins, AFB and malignant cells. Laparoscopy and biopsy is necessary when lymphoma/secondaries are suspected. CT scan and CT guided biopsy may be needed. Lymphangiography is done especially in congenital type to find out the site of leak. Medium chain fatty acids, total parenteral nutrition, operative ligation of leaking duct—are the treatment.

Chylothorax

It is accumulation of lymph in the pleural cavity. It is *common on right side* because of long course of thoracic duct towards right side. **Causes:** Injury due to trauma/surgeries in neck or chest. Surgical trauma is the *commonest* cause—may be oesophageal surgeries, pneumonectomy, cervical sympathectomy, neck dissections or aortic surgeries; tuberculosis; lymphoma or secondaries in the mediastinum;

carcinoma lung or oesophagus. **Features:** Chest pain, dyspnoea, pleural effusion; Protein loss and malnutrition; Pleural tap will show chalky white fluid rich in chylomicrons and triglyceride level > 110 mg/dl in pleural fluid is diagnostic; Chest X-ray, CT chest are needed. **Treatment:** ICT placement; TPN; often pleurodesis using bleomycin, talc, tetracycline or pleural stripping is needed; Thoracic duct ligation is beneficial in traumatic/iatrogenic cases either by open or thoracoscopic method. It is done if leak persists for more than a week.

Chyluria

It is passage of milky white chylous urine, which is aggravated after fatty meal. It may be due to obstruction in intestinal lymphatic vessels leading to high lymphatic pressure causing diversion of lymph into renal lymphatics or it may often be due to rupture of intestinal lymphatics into renal pelvis or ureter leading into lymphourinary fistula. Commonest cause is filarial. Other causes are tumour, tuberculosis, malaria and ascariasis infestation. Urinary infection, protein loss is common. It mimics bacterial/tuberculous pyuria or phosphaturia. Condition causes severe psychological and nutritional problem. Urine study, culture, IVU, lymphangiography, U/S abdomen is needed. **Treatment** is low fat protein rich diet, antibiotics, DEC, plenty of oral fluid intake, ligation of dilated lymphatics through laparotomy or sclerosing the lymph vessels.

8

Examination of Peripheral Nervous System

History taking begins with:

Name:

Address:

Age:

Sex:

Occupation:

Occupational hazard like working in lead and arsenic related industries can cause neurological problems.

History

History of Present Illness

History of Trauma

Trauma is the most common way by which a nerve is injured. Incised/penetrating/deep wounds can cause nerve injury. Sometimes fracture/dislocation can cause adjacent nerve injury. Fracture of shaft of humerus can injure radial nerve; supracondylar fracture of humerus can cause median/ulnar or radial nerve palsy; fracture of medial epicondyle of humerus can cause ulnar nerve injury; axillary nerve may be injured in subcoracoid shoulder dislocation or fracture neck of humerus; sciatic nerve (commonly common peroneal part) is injured in posterior dislocation of hip or supracondylar or subtrochanteric fractures of the femur. Fracture neck of femur may injure lateral popliteal nerve.

Traction injury can cause avulsion, neuropraxia or other types of nerve injuries causing typical lesions. Often seen in injuries to brachial plexus. Forcible increase in angle between neck and shoulder can cause injury to upper trunk of brachial plexus. During difficult labour, foetal head is pulled out with traction against shoulder causing typical upper trunk brachial plexus injury. Upper trunk lesion is called as *Erb-Duchenne palsy*. Injury to lower trunk of brachial plexus can

occur when the arm is forcibly hyperabducted causing typical *Klumpke's palsy*.

Entrapment neuropathy can cause typical nerve lesions due to compression. Tardy ulnar palsy occurs at medial epicondyle of humerus due to trapping of ulnar nerve in the callus formed after fracture of medial epicondyle and supracondylar fracture.

History of loss of sensation or loss of power: After nerve injury sensory and motor functions of the area of distribution by that particular nerve may be affected. Partial injury of a cutaneous nerve may cause intense burning pain along the distribution of nerve (*causalgia*) immediately after injury or often even many months after the healing.

Other related history: History of taking injections into the arm or thigh may cause irritation of adjacent nerve causing nerve injury. In the arm, axillary nerve may be affected causing paralysis of deltoid. In the thigh sciatic nerve may get injured by injections.

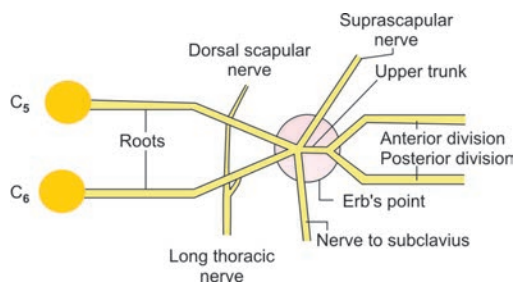
History suggestive of diabetes mellitus, alcohol intake, and leprosy are also important in nerve lesions. Earlier history of diphtheria is significant as it may cause post-diphtheric paralysis.

Local Examination

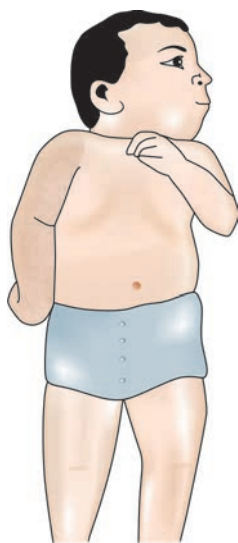
Inspection

Attitude and Deformity

Erb's palsy/Obstetrician's paralysis: 'Policeman receiving the tip' or 'Porter's tip hand' occurs in injury to upper trunk of brachial plexus (**Figs 8.1A and B**). It occurs due to traction injury; often in obstructed labour or during anaesthesia. Here junction of C₅ and C₆ is affected (Erb's point). Nerve to subclavius, supra-scapular nerve, and nerve to serratus anterior, dorsal



A



A

Figs 8.1A and B: Erb's point and Erb's palsy. Note the typical 'policeman receiving tip' sign.

scapular nerve to rhomboideus emerge close to this point. Muscles paralysed are—biceps, deltoid, brachialis, brachioradialis; partly supraspinatus, infraspinatus and supinator. Here arms hang by the side of the body adducted and medially rotated; forearm extended and pronated. There is loss of abduction and lateral rotation of the shoulder; loss of flexion and supination of forearm; absence of biceps and supinator jerks; loss of sensation over the skin over lower part of the deltoid.

Klumpke's paralysis: Here injury is to lower trunk of brachial plexus. It is due to undue abduction of the arm after fall from a height while clutching something with hands. Here C₈ and T₁ nerve roots are involved. Muscles paralysed are intrinsic muscles of hand, ulnar flexors of wrist and fingers. It causes

claw hand; cutaneous anaesthesia and analgesia along the medial border of the forearm and hand; Horner's syndrome causing ptosis, miosis, anhydrosis, enophthalmos and loss of ciliospinal reflex which is due to injury to sympathetic innervations of head and neck that leave spinal cord through T₁; vasomotor changes in the anaesthetised skin like warmth, dryness, absence of sweating and; trophic changes (**Fig. 8.2**).

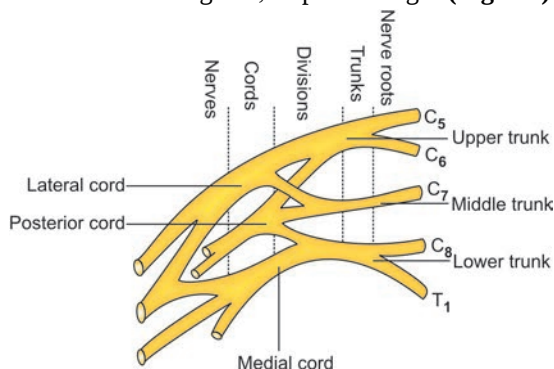


Fig. 8.2: Anatomy of brachial plexus.

Wrist drop is seen in radial nerve palsy where there is paralysis of extensor muscles; **winging of scapula** with prominent vertebral border of scapula is seen in paralysis of serratus anterior due to injury to long thoracic nerve of Bell; 'ape thumb' deformity is due to paralysis of opponens pollicis in median nerve palsy; 'pointing index' is due to paralysis of lateral half of the flexor digitorum profundus supplied by median nerve. Paralysis of dorsiflexors and evertors due to lateral popliteal nerve injury causes **foot drop** (**Figs 8.3A and B**).

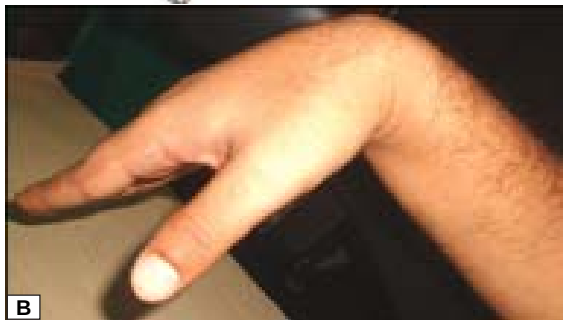
Wasting of muscles: Atrophy of particular muscles supplied by the nerve will be obvious. It is compared to opposite side in unilateral lesion. Wasting is often observed in interossei, thenar and hypothenar muscles, forearm, arm muscles, calf and thigh muscles. Muscle girth should be measured at specific point and compared to opposite side (**Fig. 8.4**).

Inspection of the Skin

Skin is inspected for dryness, glossiness, loss of skin folds and subcutaneous fat—features of paralysis. Vasomotor changes, cyanosis, excess sweat, brittle nails are observed in partial injury of the nerve.



A



B

Figs 8.3A and B: Wrist drop—due to radial nerve palsy.



Fig. 8.4: Wasting in the right hand especially over thenar eminence.

Wound if present should be inspected for its depth, site and other features. Similarly scar of old wound should be inspected. Nerve related to this wound or scar may be damaged.

Palpation

Temperature: Paralyzed limb is colder than normal.

Skin: Anaesthesia over the area of sensory supply of that particular nerve is typical. In axillary nerve injury paralysis of deltoid muscle will be present along with loss of sensation over the lower part of the deltoid. Often muscle paralysis or power cannot be assessed due to traumatic fracture of the particular site like shoulder dislocation or fracture neck of humerus in axillary nerve injury. Shifting of hyperaesthesia along the distribution of the peripheral nerve is the sign of nerve regeneration.

Wound or scar should be palpated for tenderness. Scar tenderness may signify nerve entrapment or adhesion.

Palpation of affected muscles: Paralysed muscle is soft, and flabby. It shows reduced muscle bulk and texture.

Muscle power: Muscles which are exclusively supplied by a particular nerve should be checked for altered power. Muscle power of that particular muscle is checked by the movement against resistance across the joint it acts. *Medical Research Council graded the muscle power:* 0—complete paralysis; 1—flicker of contraction; 2—contraction of muscle with gravity eliminated; 3—contractions against gravity alone; 4—contraction against gravity and some resistance alone; 5—contraction against powerful resistance.

Trapezius is checked by shrugging the shoulder against resistance. There will be wasting of trapezius with flat shoulder. It suggests spinal accessory nerve palsy. It is observed in advanced fixed neck lymph node secondaries; after radical neck dissection; trauma.

In **hypoglossal nerve palsy**, patient is asked to protrude the tongue. There will be wasting of tongue on the side of the lesion; tongue will deviate towards the same side of the injury. Hypoglossal nerve palsy occurs in advanced secondaries in neck (upper nodes); after submandibular salivary gland excision (1%); surgery to submandibular salivary gland malignancy.

Serratus anterior muscle is checked by pushing the outstretched hand against wall. Its paralysis causes prominent vertebral border and inferior angle of the scapula which will stand out of the chest wall. It is called as '*winging of scapula*'. It is due to injury to long thoracic nerve of Bell. It is derived from the C₅, C₆, C₇ nerve roots of brachial plexus. It may be injured in brachial plexus injury or chest wall/breast surgeries.

Deltoid muscle is checked with elbow flexed at right angle and abducting the arm (through shoulder joint) against resistance. Muscle contraction should be checked by palpation with the other hand.

Brachioradialis muscle is checked by asking the patient to flex the elbow against resistance keeping forearm in midprone position. It originates from upper 2/3rd of lateral supracondylar ridge of the humerus above the origin of the extensor carpi radialis longus and inserted on to the lateral side of the radius just above the styloid process. It is *flexor* of forearm in *midprone* position; supinator of fully pronated forearm. It is supplied by radial nerve C_{5, 6, 7}. It is paralysed in radial nerve injury but its action is intact in posterior interosseous nerve injury (**Fig. 8.5**).



Fig. 8.5: Checking the brachioradialis muscle power in mid-prone position. It is paralysed in radial nerve injury. It is intact in posterior interosseous nerve injury.

Extensor muscles of wrist: Extensors of the wrist are supplied by posterior interosseous nerve except extensor carpi radialis longus (supplied by radial nerve). Injury to posterior interosseous nerve will cause wrist drop with inability to extend the wrist. Wrist is extended against resistance to check the power of these muscles (**Fig. 8.6**). Patient can extend the fingers using interossei. Brachioradialis is intact in posterior interosseous nerve lesion but it will be paralysed in above elbow injury of radial nerve. Extensor carpi radialis longus, extensor carpi radialis brevis, extensor digiti minimi, extensor carpi ulnaris, extensor digitorum and anconeus are common extensors of the wrist. Extensor digitorum from its common extensor origin in the dorsum of hand divides into four slips of tendons one for each of medial four fingers. It extends into the dorsum of proximal phalanx as dorsal



Fig. 8.6: Extension of the wrist should be checked against resistance.

digital expansion. Intermediate slip attaches to middle phalanx dorsally. Two side ward tendon slips later join again to attach to dorsum of base of distal phalanx. Interossei and lumbricals pass through the tunnel to enter the dorsal expansion as wing tendons. Extensor digitorum is extensor of metacarpophalangeal joint and interphalangeal joints. Interossei and lumbrical are also flexors of the interphalangeal joints.

Flexor pollicis longus: It originates from upper 3/4th of anterior surface of shaft of radius and anterior surface of interosseous membrane; gets inserted into the palmar surface of the distal phalanx of the thumb; supplied by anterior interosseous nerve; flexor of the distal phalanx of thumb. Its power is checked by asking the patient to steady the proximal phalanx, and to bend the terminal phalanx of thumb against resistance (**Figs 8.7A and B**).



Figs 8.7A and B: Test for flexor pollicis longus. Flexion of distal phalanx of thumb against resistance is checked.

Flexor digitorum superficialis (sublimis): Its humeroulnar head originates from medial epicondyle of humerus and tubercle on the medial border of the

coronoid process and ulnar collateral ligament; radial head from anterior border of radius; it ends as four tendons one each to medial four fingers; opposite the proximal phalanx each splits into two and gets attached to medial and lateral part of the base of middle phalanx. It is the main flexor of the proximal interphalangeal joint. It is supplied by median nerve.

Flexor digitorum profundus (FDP): It originates from upper 3/4th of anterior and medial surface of ulnar shaft, olecranon and coronoid process of ulna and anterior surface of the interosseous membrane; it ends as 4 tendons one for each medial 4 fingers; each after passing through the split sublimis attaches to base of the distal phalanx in front. It is the chief flexor of the distal phalanx. It is a composite hybrid muscle wherein medial two tendons are supplied by ulnar nerve and lateral two tendons are supplied by anterior interosseous nerve. Both sublimis and FDP has got synovial folds called as *vincula longa* and *vincula brevia*.

Ochsner's clasp test: If the patient is asked to clasp the hands, index finger of the affected side fails to flex and remains as *pointing index*. It suggests median nerve injury (**Figs 8.8A and B**).



Figs 8.8A and B: Ochsner's clasp test: If the patient is asked to clasp the hands, index finger of the affected side fails to flex and remains as pointing index. It suggests median nerve injury.

Abductor pollicis brevis: It originates from the tubercle of the scaphoid, crest of trapezium, flexor retinaculum; gets inserted to lateral side of base of proximal phalanx of thumb. Its nerve supply is median nerve (C_8, T_1). It abducts the thumb at metacar-

pophalangeal joint and carpometacarpal joints with associated medial rotation. Its power is tested by moving the thumb upwards at right angle to the palm of the hand with palm laid flat on the table. Patient is asked to keep his hand flat supine on the table. A pen tip is kept near thumb in front at higher level; patient is asked to abduct his thumb to touch the pen held. In normal functioning muscle patient can touch the pen otherwise he cannot. It is called as '*pen test*' (**Figs 8.9 and 8.10**).



Fig. 8.9: Pen test for abductor pollicis brevis—median nerve.

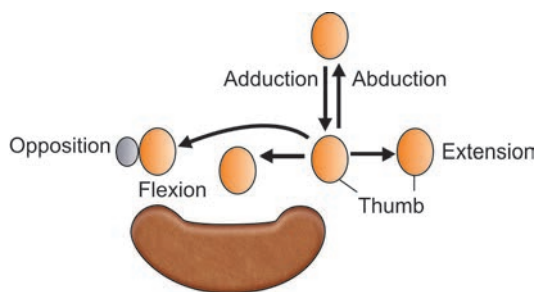


Fig. 8.10: Abduction and adduction of thumb occurs in right angle to the plane of flexion and extension of thumb.

Opponens pollicis: It originates from crest of trapezium and flexor retinaculum; gets inserted to lateral half of the palmar surface of the first metacarpal bone; supplied by median nerve (C_8, T_1). It causes opposition of thumb with combination of flexion and medial rotation. It is checked by swinging the thumb across the palm to touch tips of other fingers (**Fig. 8.11**).



Fig. 8.11: Checking opponens pollicis against resistance.

Flexor carpi ulnaris: Its origins is from common flexor origin –humeral head from medial epicondyle; ulnar head from the medial margin of olecranon. It is inserted into the pisiform bone, base of the 5th metacarpal bone, hook of the hamate as pisometacarpal and pisohamate ligaments. It is supplied by ulnar nerve. Its action is flexion of wrist, adduction of wrist. Ulnar nerve passes between two heads of this muscle. Ulnar vessels and nerve are lateral its tendon just above the wrist. Pisiform is a sesamoid bone of this muscle. Its power is checked by flexing the wrist against resistance and deviation of hand towards radial side is seen due to defective wrist adduction (**Fig. 8.12**).

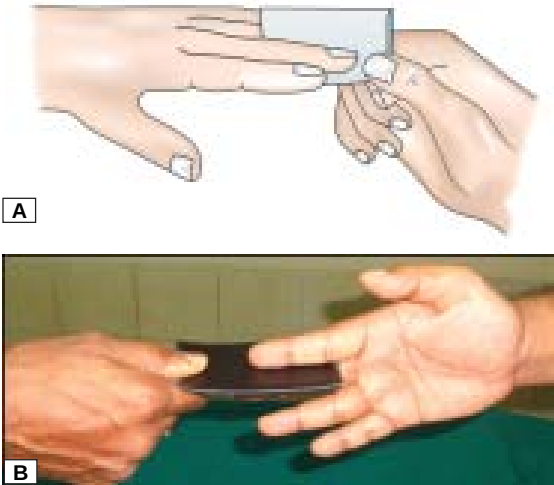


Fig. 8.12: Flexion of the wrist should be checked against resistance.

Lumbrical muscles: They are four small muscles originating from tendons of the flexor digitorum profundus numbering of which is done from lateral to medial—1, 2, 3 and 4. Their origins are shown in **Figure 8.15**. They are inserted into the 2nd, 3rd, 4th and 5th dorsal digital expansions of the proximal phalanges on their lateral sides. 1st and 2nd lumbricals are supplied by median nerve (C_8, T_1); 3rd and 4th lumbricals are supplied by deep branch of ulnar nerve (C_8, T_1). Along with interossei they extend to proximal and distal interphalangeal joints; and also flex the metacarpophalangeal joints.

Palmar interossei: They are 4 small muscles between metacarpals numbered as 1st, 2nd, 3rd and 4th from lateral to medial. 1st muscle originates from *medial side* of the base of 1st metacarpal bone and gets inserted to medial side of the proximal phalanx of thumb. 2nd muscle originates from *medial side* of shaft of 2nd metacarpal, gets inserted to medial side of proximal phalanx and dorsal digital expansion of index finger. 3rd muscle has got its origin from *lateral part* of the shaft of the 4th metacarpal inserting into the base of proximal phalanx and dorsal digital expansion of the ring (4th) finger. 4th muscle begins from *lateral part* of the shaft of 5th metacarpal gets inserted into proximal phalanx and dorsal expansion of little (5th) finger. There is no palmar interosseous to middle finger. It is supplied by deep branch of the ulnar nerve (C_8, T_1). **Actions:** All palmar interossei adduct the finger (*PAD*) with middle finger as the centre line. They also flex the metacarpophalangeal joint and extend the interphalangeal joints along with lumbricals (**Figs 8.13A to 8.14B**).

Dorsal interossei: They are 4 small muscles between metacarpals numbered from lateral to medial. 1st originates from shafts of 1st and 2nd metacarpals; 2nd from shafts of 2nd and 3rd bones; 3rd from shafts of 3rd and 4th bones; 4th from shafts of 4th and 5th bones. First is inserted to lateral aspect of dorsal digital expansion and base of proximal phalanx of index finger; second to lateral aspect of middle finger; third to medial aspect of middle finger; fourth to medial aspect of ring finger. Dorsal interossei is not inserted to thumb and little fingers. Middle finger has got two dorsal interossei insertions on either side. Dorsal interossei are supplied by deep branch of ulnar nerve



Figs 8.13A and B: Card test: A card is placed between the two fingers of the patient to grasp. In weak palmar interossei, patient cannot grasp (palmar interossei are adductors of the fingers—PAD).

(C₈, T₁). **Actions:** They are abductors (*DAB*) of the fingers with middle finger as centre line of action. Thumb and little finger has got their own abductors and so they do not need dorsal interossei. Abduction of fingers occurs in the plane of the palm whereas abduction of thumb occurs in a plane right angle to the plane of the palm (**Figs 8.15 and 8.16**).

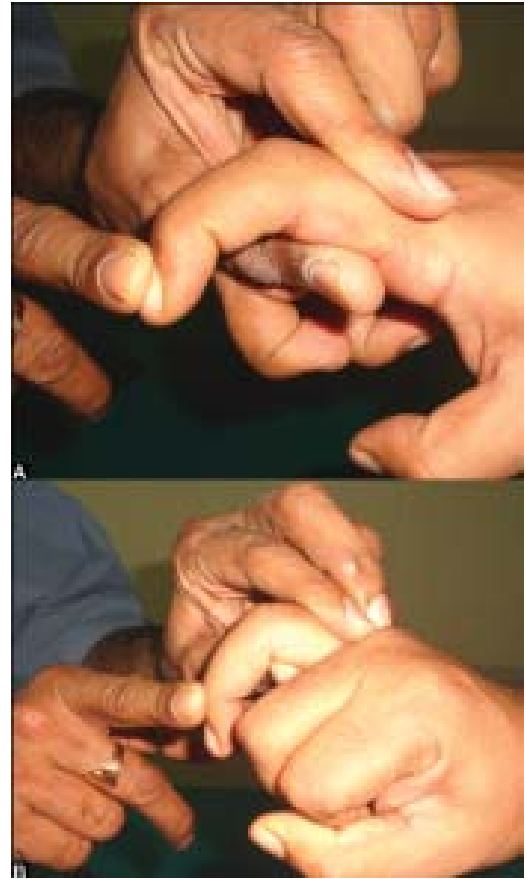


Fig. 8.14: Extension of interphalangeal joint should be checked against resistance by fixing.

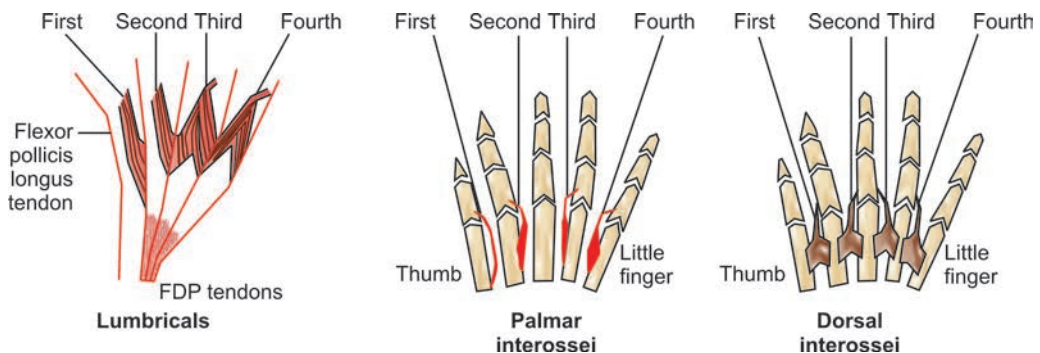


Fig. 8.15: Figure showing attachments of lumbricals and interossei. Lumbricals after origin from FDP tendons get inserted to lateral aspects of extensor hoods of medial four fingers. 1st and 2nd palmar interossei are attached to medial aspect of the proximal phalanx of thumb and index; 3rd and 4th are inserted into the lateral side of the ring and little fingers; no palmar interossei is attached to middle finger. 1st and 2nd dorsal interossei are inserted to lateral aspects of base and dorsal expansion of proximal phalanges of index and middle fingers. 3rd and 4th interossei are inserted to medial aspect of proximal phalanges of middle and ring fingers. Middle finger has got on either sides insertions of 2nd and 3rd dorsal interossei.

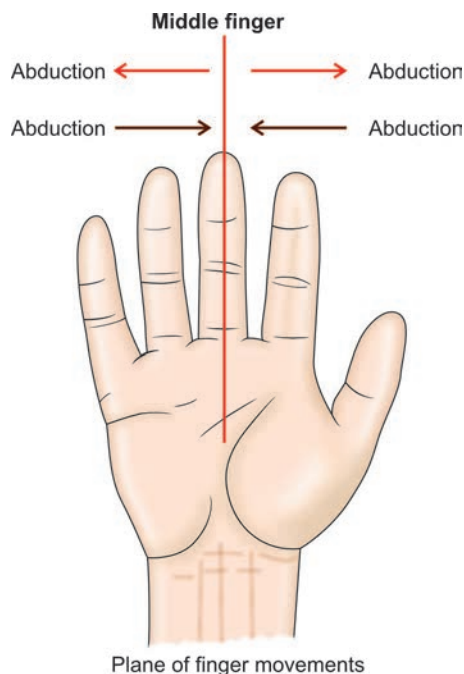


Fig. 8.16: Plane of finger movements is along the middle finger. Palmar interossei are adductors of the fingers (PAD); dorsal interossei are abductors of the fingers (DAB).

Adductor pollicis: Oblique head has origin from capitate bone and bases of 2nd and 3rd metacarpal bones; transverse head from palmar part of 3rd metacarpal bone. It is inserted into medial side of the base of the thumb. It is supplied by deep branch of ulnar nerve (C_8, T_1). It adducts the thumb from abducted or flexed position assisted by first palmar interossei. It helps in forceful gripping. Patient is given a book to hold between extended thumb and fingers. If ulnar nerve is normal, he can hold the book with extended thumb using adductor pollicis and first palmar interossei. If there is ulnar paralysis, grip on book is assisted by flexing the terminal phalanx using flexor pollicis longus (supplied by median nerve). This book test is called as *Froment's sign*. It can also be confirmed by holding the card firmly between extended thumb and other fingers – *card test* (Figs 8.17A and B).

Sciatic nerve injury is rare. When it develops it is complete paralysis of the *hamstring muscles* and muscles below the knee. Incomplete lesion commonly involves common peroneal nerve (lateral popliteal



Figs 8.17A and B: Froment's test and card test for ulnar nerve—adductor pollicis. Thumb has to be flexed in paralysed adductor pollicis by over action of flexor pollicis longus to have grip on book or card.

nerve). There is paralysis of extensor and peroneal muscles of the leg causing *talipes equino varus* and inability to dorsiflex and evert the foot with undue lifting of the foot to clear 'dropped foot' high from the ground – 'foot drop'. Tibial nerve supplies the plantar flexors of the ankle joint. Patient will not be able to plantar flex the ankle joint causing '*talipes calcaneo valgus*' (Fig. 8.18).

Sensation: Light touch, pressure, localisation, two point discrimination, pain, temperature, sense of position, size, shape and form of the object recognition, vibration sense—all should be checked in nerve injury/diseases. Sensations are checked from *impaired area towards normal area*. *Light touch* is epicritic sensation used to locate accurate area of loss of sensation. It is done using cotton. *Gross touch* is protopathic sensation which is checked by fingertip. Sensation



Fig. 8.18: Foot drop with claw toes. It is due to peroneal nerve injury (Lateral popliteal nerve).

of two point discrimination is checked using a compass points. A 2 mm separation can be made out normally which is impaired in a nerve injury. It is transmitted by posterior column of spinal cord. *Superficial pain* sensation from the skin is elicited using sharp pin. *Deep muscular or bone pain* is elicited by gentle pressure or squeezing. *Temperature* is checked using warm and cold water in test tubes. Inability to recognise the size, shape and form of the object is called as *astereognosis*. *Position sense* is joint's spatial orientation. It is usually checked in great toe or other toes; thumb or other fingers. Position sense is checked with patient eyes closed and eliciting the joint movements passively by holding its outer aspect (laterally); and the patient is asked which position the joint is held. Position sense is often lost with astereognosis in posterior column lesions. Only astereognosis with normal position sense and light touch is seen in parietal lobe injury. *Vibration sense* is checked using tuning fork 128 Hz by placing over the surface (bony protuberance). Vibration sense is lost in tabes dorsalis, peripheral neuritis, and posterior column disorders (**Figs 8.19A to 8.20D**).

Reflexes like biceps, supinator, triceps, knee, plantar, ankle should be checked for changes. Reflexes

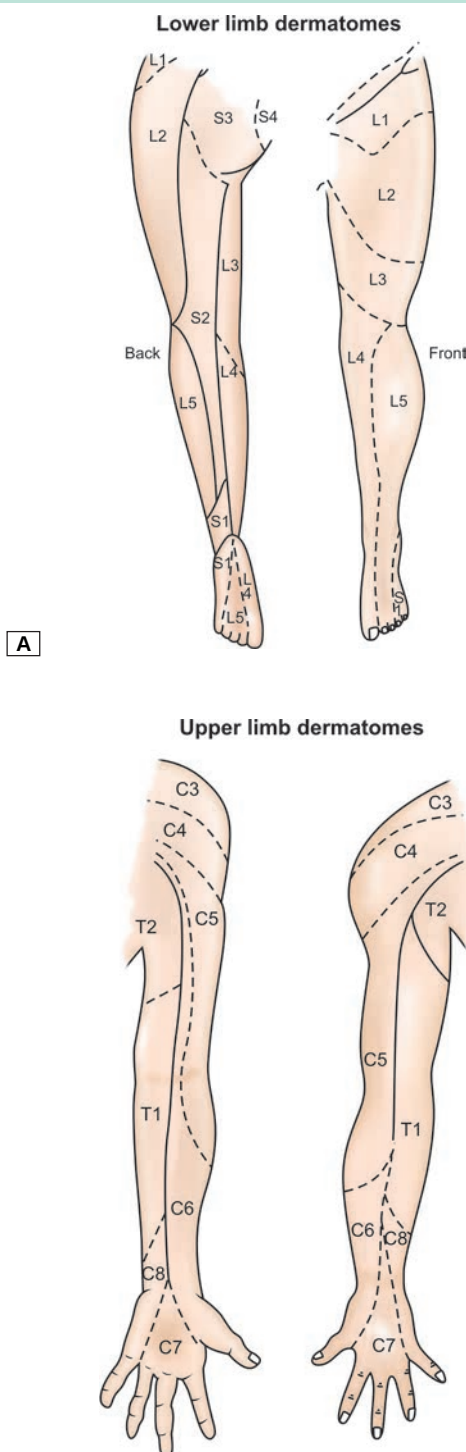
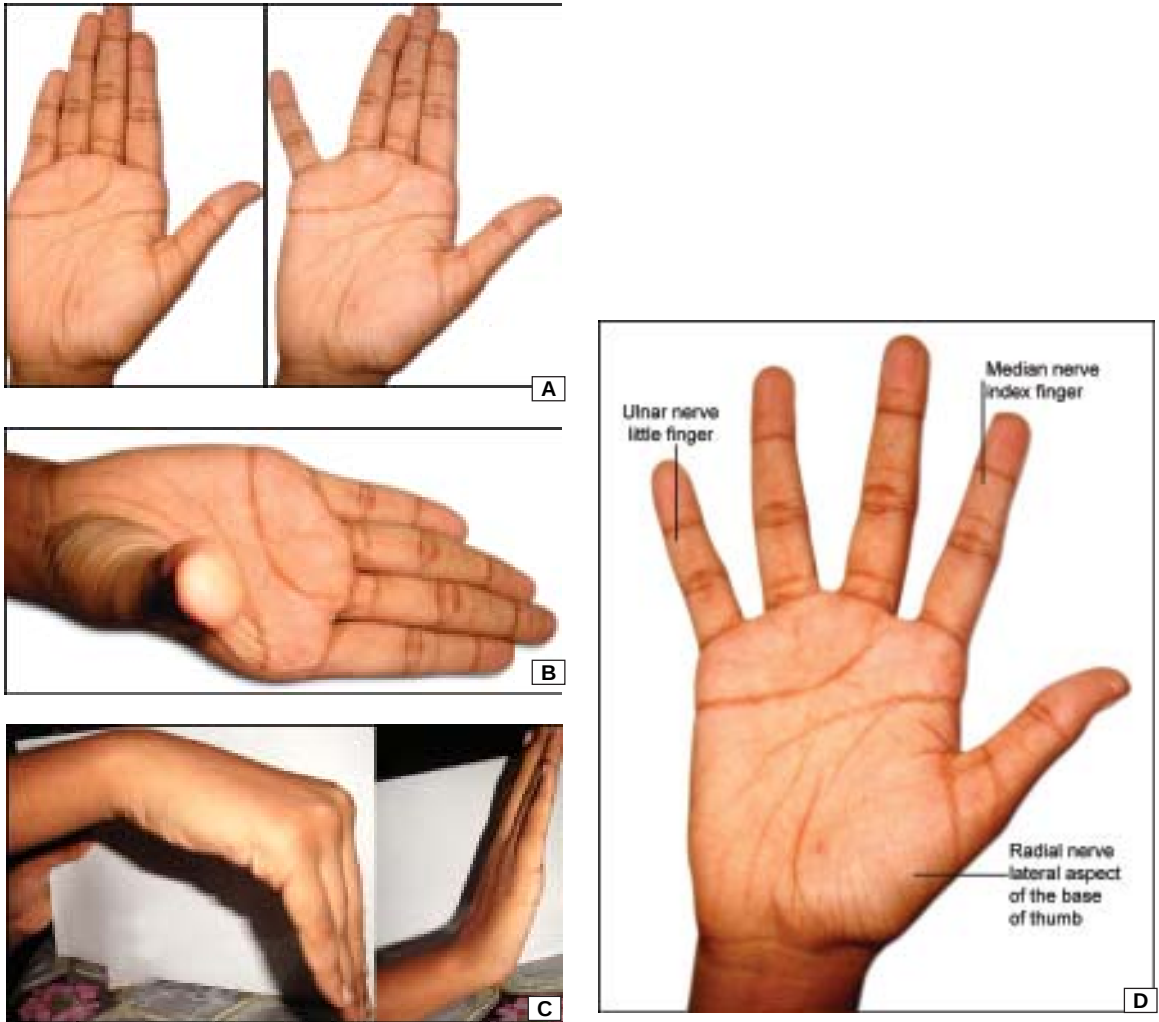


Fig. 8.19: Upper limb and lower limb dermatomes.



Figs 8.20A to D: Quick tests for motor and sensory nerves in the hand – ulnar, median and radial nerves. A,B,C—Motor, D—Sensory

are *stretch reflexes* and are indicators of the integrity of the spinal segments. Tendon is stretched using a rubber hammer. Often patient is asked to clench the teeth or interlock fingers so that site to be tested is relaxed properly. Biceps – C_{5,6}; Triceps – C_{6,7}; Finger jerk – C₈; Supinator – C₈; Knee jerk – L_{2,3,4}; Ankle jerk – S_{1,2}; Plantar reflex – L₅, S₁, S₂; Abdominal reflexes – T₈, T₉, T₁₀ and T_{10,11}; Cremasteric reflex – L₁ segment. In plantar reflex, lateral aspect of the sole of the foot when scraped causes a withdrawal reflex and flexion of the great toe. Great toe extension

(upward) occurs in upper motor neuron lesion. Abdominal reflexes are elicited by stroking upper and lower abdomen which causes contraction of rectus abdominis muscle. In cremasteric reflex inner side of the thigh is stroked to contract cremaster muscle.

Movements of the related joint should be checked. Both active and passive movements should be checked. In paralysed muscle passive movements are increased more than active movements (Normally passive and active movements are near equal; passive movement

is elicited by the examiner; active movement is done by the patient himself).

Palpation of area of deformity and confirming it is also necessary.

Palpation of injured area; scar; peripheral pulses; regional lymph nodes should be done. Nerve thickening, sensation over a skin patch may need to check in case of leprosy. Gentle tapping over the course of the peripheral nerve is done to elicit hyperaesthesia or 'pins and needles' which is a sign of regeneration of injured nerve—*Tinel's sign*.

Systemic Examination

Examination of respiratory system; examination in relation to features of alcoholism, diabetic neuropathy, neuritis, syphilis, beriberi, lead poison, arsenic poison are essential.

Examination of spinal cord and central nervous system is essential.

Relevant Investigations

Blood tests for diabetes; peripheral smear; haemoglobin. Urine analysis.

Nasal scraping, skin biopsy, nerve biopsy for leprosy. Usually *sural nerve biopsy* is done. It is done under local anaesthesia by making incision over the lateral aspect of the leg or adjacent to lateral malleolus. Nerve abscess and AFB staining will confirm the Hansen's disease.

Nerve conduction study: It is demonstration of nerve potentials. It is used to find out nerve regeneration. It is useful to differentiate from cervical spondylosis, carpal tunnel syndrome or cervical rib syndrome with neurological manifestations.

Electrical stimulation: It is to assess reaction of degeneration. It begins in 4th day of nerve injury and establishes in 2 weeks. It is seen in denervated muscle. Normally cathodal/kathodal closure contraction (KCC) is stronger than anodal closure contraction (ACC). In muscle denervation, there is no response to Faradic stimulation but weak galvanic response on reverse – Anodal closure contraction has become stronger than Kathodal closure contraction.

Other relevant investigations related to cause—MRI, serum tests, etc.

Peripheral Nerve Injuries

Figure 8.21 shows cross-section view of a peripheral nerve.

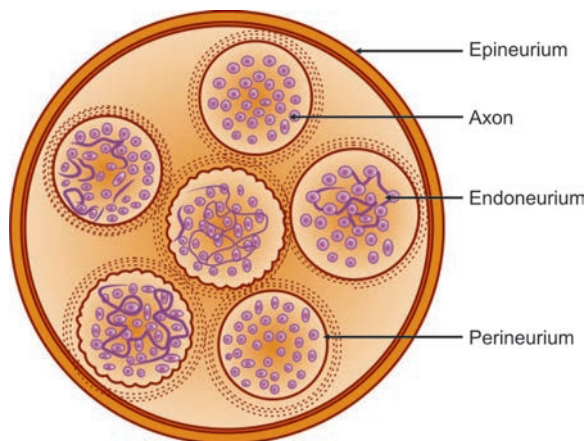


Fig. 8.21: Cross-section of a nerve.

Seddon's Classification

Neuropraxia: It is temporary physiological paralysis of nerve conduction. Here recovery is complete in few hours to weeks. There is no reaction of degeneration. It is due to stretching/torsion/transmitted injury.

Axonotmesis: It is division of nerve fibres or axons with intact nerve sheath. It is an incomplete nerve injury. Wallerian degeneration occurs distally. It is due to stress/compression/traction by fractures/dislocations/exercises. There is reaction of degeneration distally with anodal closure contraction greater than cathodal closure contraction with near complete recovery. Position of axons and nerve is intact. Patient can present with sensory loss, paralysis of muscles or causalgia. There is loss of sensation, loss of muscle tone and power, reduced reflex. Total area affected is lesser than the area supplied by the affected nerve. Disused atrophy of the affected area occurs with thin skin, brittle nails, cold and blue tissues.

Neurotmesis: Here complete division of nerve fibres with sheath occurs. Degeneration occurs proximally up to the first node of Ranvier (retrograde degeneration) as well as distal to the injury. Recovery is incomplete even after nerve suturing. There is complete loss of motor and sensory functions with loss of reflexes.

Recovery is still poorer if the nerve is of mixed type other than pure motor or sensory type. Example is recovery of radial nerve injury at elbow is better than recovery of median or ulnar nerves.

Injuries may be incised or lacerated or crushed one. Cut end of the nerve forms proximally *neuroma* and distally *glioma*.

Neuromas may be—True neuroma or False neuroma; *End neuroma* or *Side neuroma*.

Sunderland's Classification

- I. Conduction block—temporary neuronal block.
- II. Axonotmesis but endoneurium is preserved.
- III. Axonotmesis with disruption of endoneurium but perineurium is preserved.
- IV. Here there is disruption of endo and perineurium but epineurium is intact.
- V. Neurotmesis with disruption of endo, peri and epineurium.

Clinical Features

Loss of sensory, motor, autonomous and reflex functions; secondary changes in the skin and joints.

Prognostic Factors

Prognostic factors in healing of the nerve injury: Higher the lesion worse the prognosis; more the gap between the cut ends worse the prognosis; associated injuries alter the prognosis; children do better with nerve injury; type of the injury also decides the prognosis.

TINEL'S SIGN

It is the clinical sign (prognostic indicator) used to assess the level of regeneration. It is elicited 3 weeks after the nerve injury (Regeneration begins after the

completion of nerve degeneration). It is done by tapping over the course of the nerve *from distal to proximal* to elicit a sensation of '*pins and needles*' or *hyperaesthesia*. If sensation is felt at the site as well as distally along the distribution of the nerve that means *good recovery* can be expected. If sensation is felt only at the site of tapping, then result is *equivocal*. If no sensation is felt it means *no recovery*.

Causes of Peripheral Nerve Lesions

Nerve injury/disease may be single nerve disease or multiple nerve diseases.

Traumatic: Either closed or open injury.

Inflammatory: Leprosy, herpes zoster, diphtheria.

Compression neuropathies.

Lead and arsenic poisoning.

Alcoholism.

Metabolic: Diabetes mellitus, B₁ deficiency (Beriberi), Porphyria.

Neurofibroma and other neural tumours.

Idiopathic.

Brachial Plexus Injury

It can be—Supraclavicular injury—65%; Infraclavicular injury—25%; Combined—10%.

It can be

- **Pre-ganglionic injury** like avulsion injury; more dangerous; extends into the spinal cord.
- **Post-ganglionic injury**—usually less severe; better recovery.

Causalgia

It is severe burning pain in the distribution of a peripheral nerve due to *incomplete injury* to the peripheral nerve. *Sites:* Common in upper limb, seen

Upper plexus injury (Erb-Duchenne paralysis)	Lower plexus injury: (Klumpke's paralysis)
It is due to depression of shoulder by trauma After difficult labour in newborn Muscles affected are: Deltoid, Biceps, Brachioradialis, Supinator. Elbow will be extended; pronated and upper limb is internally rotated (<i>Policeman receiving tip</i>). Sensory deficit over the lateral aspect of arm and upper part of the lateral forearm.	Forcible hyperabduction causes this injury. In newborn due to difficult breech delivery Here C ₈ and T ₁ are injured Intrinsic muscles of the hand are involved <i>Effects are:</i> Combined median and ulnar claw hand Horner's syndrome Sensory deficit over the medial aspect of the forearm, hand and medial 1½ finger

in brachial plexus or median nerve injuries. In the lower limb it can be seen in sciatic nerve or tibial nerve distribution. *Clinically*, there will be hyperaesthesia and severe, disabling burning pain along the distribution of the nerve.

Median Nerve Injury

Median nerve arises from lateral ($C_{5,6,7}$) and medial cord (C_8 and T_1) of the brachial plexus. It is initially lateral to the axillary artery and becomes medial in the lower part of the arm and in the cubital fossa. It passes through the two heads of the pronator teres, descends in relation to flexor muscles (deep to flexor digitorum superficialis and superficial to flexor digitorum profundus); later over the lateral edge of the flexor digitorum superficialis and between it and flexor carpi radialis. It enters the palm through the carpal tunnel at the wrist. It supplies pronator teres, flexor carpi radialis, palmaris longus and flexor digitorum superficialis. Anterior interosseous branch of the median nerve supplies pronator teres, lateral half of the flexor digitorum profundus, flexor pollicis longus and pronator quadratus. In the wrist, it supplies abductor pollicis brevis, flexor pollicis and opponens pollicis of thenar eminence and lateral two lumbricals. It gives sensory supply to lateral three and half fingers of the hand.

Median nerve is affected in: 1. Injuries - Supracondylar fracture of the elbow; Fracture-dislocation of the elbow; Direct cut injuries. 2. Leprosy; 3. Carpal tunnel syndrome; 4. As a part of brachial plexus injury.

Clinical features of median nerve palsy: In high median nerve palsy—Wasting of the thenar eminence; loss of sensation in lateral three and half fingers; *Ochsner's clasping test* shows *pointing index* because of the inactivity of flexor digitorum superficialis and lateral two divisions of the profundus; '*Ape or Simian thumb deformity*' is due to overaction of the adductor pollicis which is supplied by the deep branch of ulnar nerve. As all other thenar muscles are paralysed, thumb comes in the same plane of the metacarpals. '*Pen test*': In median nerve injury, pen held in front of the hand cannot be touched by thumb as abduction is not possible due to paralysis of the abductor pollicis brevis. Flexor carpi radialis is paralysed and so hand deviates towards

ulnar side when flexed against resistance. In low median nerve palsy- *Flexor digitorum profundus* is not paralysed and so *pointing index* is not seen. Loss of action of opponens pollicis is seen (**Figs 8.22 and 8.23**).

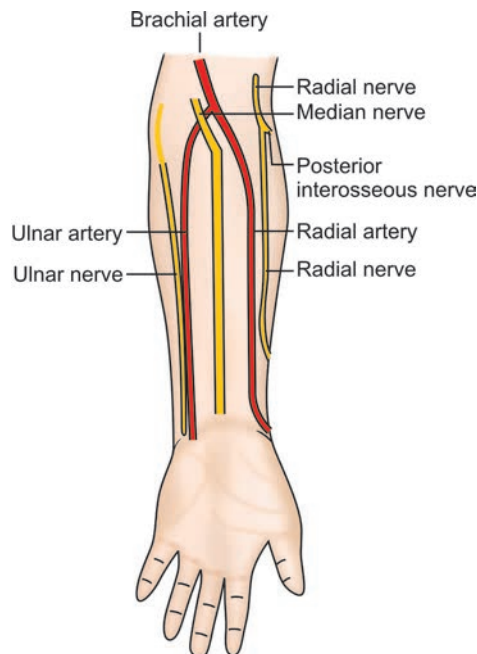


Fig. 8.22: Nerves of forearm – ulnar, median, radial and their branches.

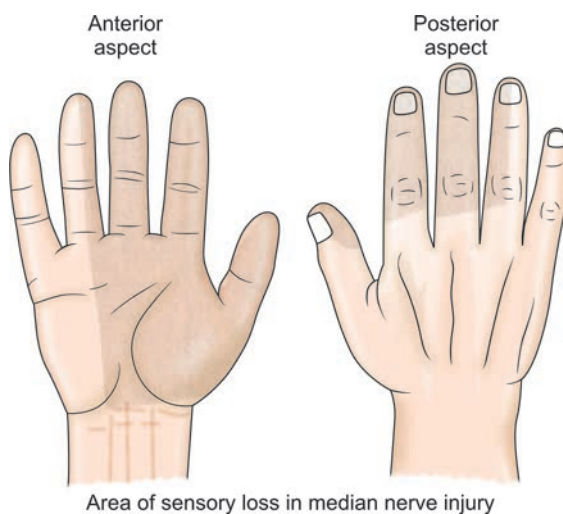


Fig. 8.23: Sensory loss in median nerve injury.

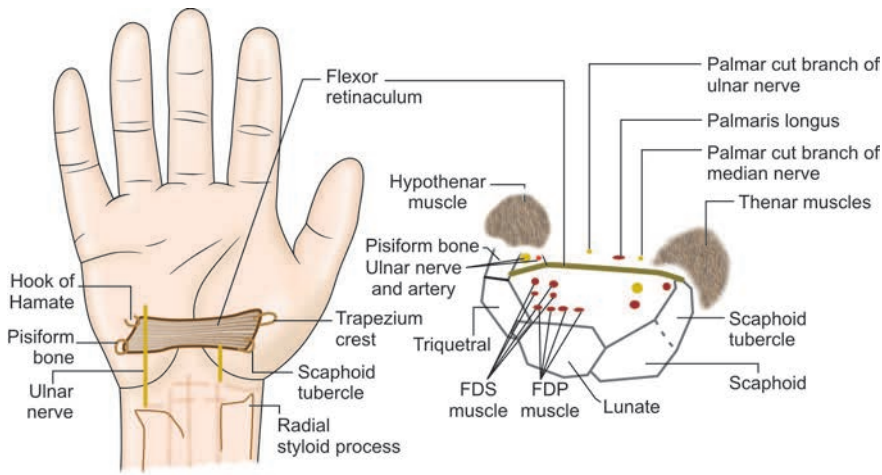


Fig. 8.24: Anatomy of carpal tunnel.

Carpal Tunnel Syndrome

It is the compression neuropathy of median nerve in the carpus, deep to flexor retinaculum (osseo fibrous tunnel/canal). It is actually a stenosing tenosynovitis/tenovaginitis under the flexor retinaculum. Flexor retinaculum (transverse carpal ligament) maintains the concavity of wrist and extends laterally from trapezium and scaphoid to pisiform and hook of the hamate medially. *Carpal tunnel* is formed by carpal bones behind and flexor retinaculum in front. It contains median nerve and long flexor tendons of fingers and thumb. Ulnar nerve lies superficially, not in the carpal tunnel. Median nerve gets compressed if space of the carpal tunnel gets reduced (**Fig. 8.24**).

Causes—Lunate dislocation; malunited Colles' fracture; radiocarpal arthritis; flexor tendon tenosynovitis; myxoedema; acromegaly; pregnancy.

Clinical features—Common in *females*; where there is tingling, numbness, paraesthesia and burning sensation in the lateral three and half fingers supplied by median nerve; burning sensation gets aggravated at night and Ape thumb deformity, wasting of thenar muscles, weakness of opponens pollicis and abductor pollicis brevis are other features of low median nerve palsy. When BP cuff is inflated patient feels the typical pain in the fingers; tapping the median nerve at the distal end of forearm with the wrist held in extension aggravates the symptoms; condition is often bilateral. There is also difficulty in flexing the fingers with pain.

Often mild tenderness over the flexor retinaculum may be elicited. Symptoms exacerbate at night typically. Pressure on the retinaculum does not induce the symptoms; but flexing the wrist fully and holding in that position for 2 minutes produce symptoms. Light touch and two point discrimination is affected in the $3\frac{1}{2}$ fingers and palm. There is loss of muscle bulk of thenar eminence which is easily felt when these muscles are contracted. There are no features of arterial insufficiency (Refer Chapter 30, pg 686).

Differential diagnosis—Cervical spondylosis; cervical rib syndrome. Diagnosis is by nerve conduction studies.

Ulnar Nerve Injury

After arising from the medial cord of the brachial plexus (C_8 and T_1), it runs on the medial aspect of the axillary artery up to middle of the arm. Then it enters the posterior compartment in relation to triceps muscle. After passing behind the medial epicondyle along with superior ulnar collateral artery and through two heads of flexor carpi ulnaris, it runs in front of the flexor digitorum profundus (FDP) in the forearm. It reaches the hand in front of the flexor retinaculum through *Guyon's canal*. It stays between pisiform bone medially and ulnar artery laterally. Here it divides into superficial and deep branches.

Ulnar nerve supplies flexor carpi ulnaris, medial half of flexor digitorum profundus, all muscles of the

hypotenar eminence (palmaris brevis, abductor digiti minimi, opponens digiti minimi, and flexor digiti minimi), and adductor pollicis of the thenar eminence, 3rd and 4th lumbricals and all interossei of the hand. It also gives sensory supply to medial part of the hand, medial one and half fingers. *Ulnar nerve is affected in:* Supracondylar fracture; injury to the medial epicondyle; tardy ulnar palsy (entrapment neuropathy behind the medial epicondyle); leprosy; cubitus valgus deformity.

Clinical Features

Claw hand deformity along with weakness of all the muscles supplied by the ulnar nerve is seen; *Card test:* A card is placed between the two fingers of the patient to grasp. In weak palmar interossei, patient can not grasp (palmar interossei are adductors of the fingers—PAD). Abduction of fingers is also checked (dorsal interossei are abductors) (DAB). *Froment's sign:* A book is placed to grasp between fingers and thumb of the patient. Normally thumb will be straight because of the action of adductor pollicis muscle. Because it is paralysed in ulnar palsy, grasp is achieved by action of flexor pollicis longus and there will be flexed thumb. Loss of sensation over medial one and half fingers and hand is seen (**Fig. 8.25**).

Intrinsic minus deformity: It is due to loss of intrinsic muscle power, i.e. claw hand.

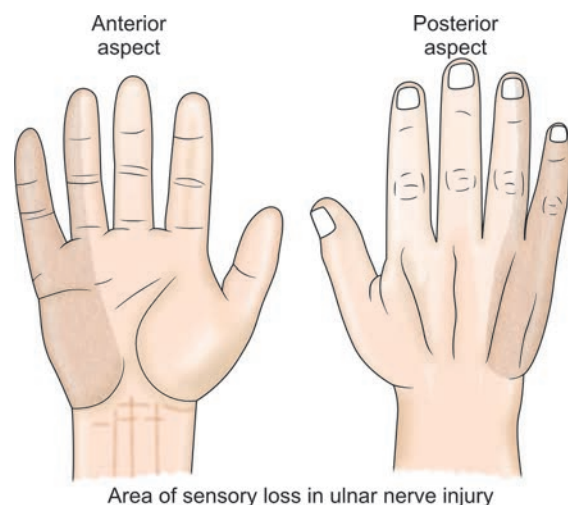


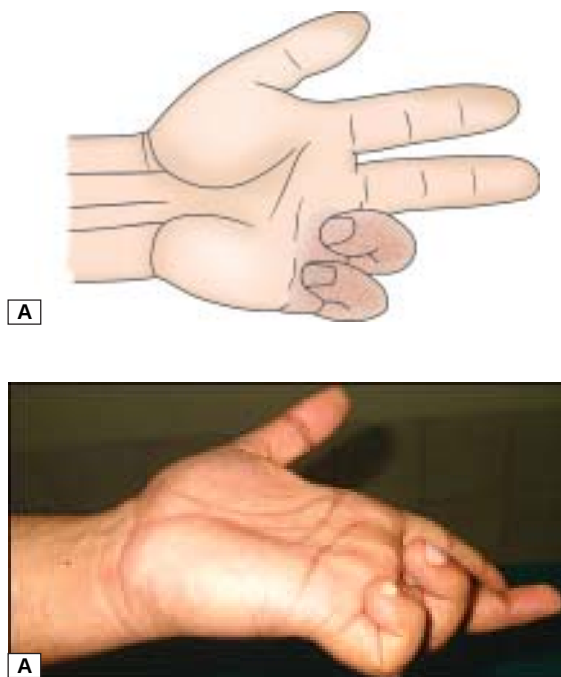
Fig. 8.25: Sensory loss in ulnar nerve injury.

Intrinsic plus deformity: It is due to muscle contracture and fibrosis.

Ulnar paradox: In ulnar palsy higher the lesions, lesser the deformity, lower the lesion, more the deformity. In higher lesion, FDP is also paralysed. In lower lesion FDP is intact and so FDP causes more flexion (over action) and so aggravates the claw hand.

Claw Hand

It is the hyperextension of the metacarpophalangeal joint with flexion of the interphalangeal joints of the hand. Extension of MCP joint is due to action of extensor digitorum; Flexion of MCP joint and extension of interphalangeal joints are by (through extensor hood) interossei and lumbricals (Main en griffe). So extensor hood is functioned by ulnar nerve mainly and also by median nerve (**Figs 8.26A and B**). In ulnar or median nerve palsies, these actions are paralysed and so patient develops claw hand. It is actually *intrinsic minus deformity*.



Figs 8.26A and B: Ulner claw hand with hyperextension of metacarpophalangeal joints and flexion of proximal and distal interphalangeal joints in medial two fingers due to ulnar nerve palsy.

Causes: Leprosy; trauma; entrapment neuropathies; tardy ulnar palsy; Klumpke's palsy and rare causes (like syringomyelia, poliomyelitis, amyotrophic lateral sclerosis, Volkmann's contracture). Causes may be neurological or musculoskeletal. Claw hand is a deformity occurring due to loss of motor function; but often there will be associated sensory loss also.

Clinical Features: Typical claw hand; Loss of sensation along the distribution of the nerve; inability to grasp card between the fingers; while holding the book between the thumb and fingers, thumb will be flexed in ulnar claw hand (Froment's test).

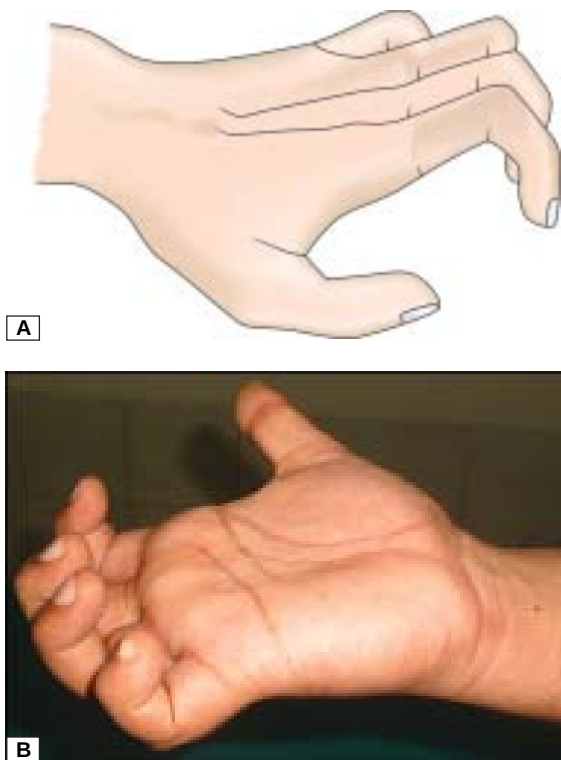
Types: 1. **Ulnar claw hand:** Only medial two fingers are involved. a) **Low ulnar palsy:** Here lesion is in the wrist (at Guyon's canal). Here deformity is more because of the over action of the FDP. b) **High ulnar palsy:** Here FDP is also paralysed and over action is not there. So deformity is lesser. **Ulnar paradox:** Higher the lesion lesser the deformity, lower the lesion

more the deformity. 2. **Median claw hand:** Only lateral two fingers are involved. It is less common. 3. **Combined median and ulnar claw hand:** Here all four fingers of the hand are involved (**Figs 8.27A and B**).

Radial Nerve Lesions

Radial nerve is derived from the posterior cord of the brachial plexus ($C_{5,6,7,8}$ and T_1). It descends behind the axillary artery in front of the subscapularis, latissimus dorsi and teres major. It passes through the medial and lateral heads of the triceps muscle, winds round the humerus through the radial groove and enters the forearm in front of the lateral epicondyle and in relation to brachioradialis, brachialis and extensor carpi radialis longus muscles. In the arm it supplies triceps, anconeus, brachioradialis, extensor carpi radialis longus and part of the brachialis. It gives posterior and lower lateral cutaneous nerves of the arm and posterior cutaneous nerve of the forearm. **Superficial branch** of the radial nerve from the elbow runs in the forearm in relation to supinator and brachioradialis and ends by forming 5 digital nerves which gives sensory supply to lateral side of the thumb, related part of thenar eminence, three and half fingers on the dorsal aspect (upto the root of the nail, upto the middle phalanx of the index finger, upto the proximal interphalangeal joints of the middle and ring fingers). **Deep branch** also called as posterior interosseous nerve winds round the radius supplying supinator and extensor carpi radialis brevis. It gives 3 short branches to extensor digitorum, extensor digiti minimi and extensor carpi ulnaris. It also gives two long branches (One to abductor pollicis longus and extensor pollicis brevis; another extensor pollicis longus and extensor indicis).

Conditions where radial nerve is affected: In the axilla—**Crutch palsy:** It is neuropraxia; due to fracture upper end of the humerus; bony or soft tissue growth. **In the radial groove**—Pressure on the arm from the edge of the operating table; **Saturday night palsy:** An individual with excessive alcohol consumption compresses his arm over the chair or by fall. It is neuropraxia. Prolonged tourniquet application—**tourniquet palsy.** Fracture of the shaft of the humerus. Rarely intramuscular injection of drugs can cause



Figs 8.27A and B: Combined claw hand involving all fingers due to both ulnar and median nerve injuries.

radial nerve palsy. *In the elbow* – due to dislocation or fracture neck of the radius.

Clinical features: *Wrist drop* due to inability to extend the wrist; inability to extend metacarpophalangeal joint, but extensions of the interphalangeal joints are normal; inability to extend the forearm; inability to extend the thumb; flexion of the elbow against resistance with forearm in mid-prone position is difficult because of the weakness of the brachioradialis muscle; loss of sensation in back of the arm, forearm, hand and lateral three and half fingers (**Fig. 8.28**).

Posterior interosseous nerve is purely motor and so when it gets injured the sensation is intact. Brachioradialis is supplied by radial nerve not by posterior interosseous nerve and so its action is intact which can be confirmed by checking against resistance.

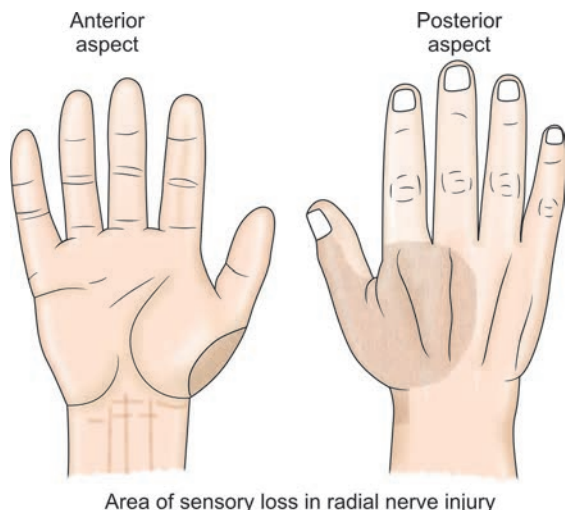


Fig. 8.28: Sensory loss in radial nerve injury.

Common Peroneal Nerve

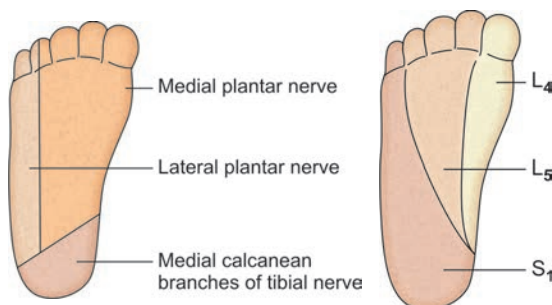
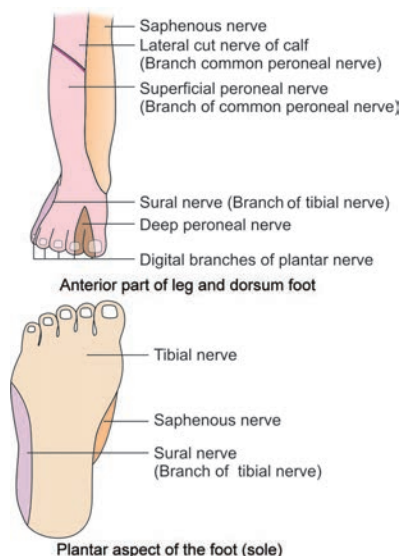
This nerve supplies the extensor and peroneal group of muscles and sensory supply to the skin over the front and lateral aspect of the leg and dorsum of the foot.

Common peroneal nerve is affected by—fracture neck of the fibula; leprosy; lead poisoning; iatrogenic.

Clinical features: Foot drop with high stepping gait; talipes equino-varus deformity; loss of sensation in the lateral side of the leg and dorsum of the foot (**Figs 8.29A to 8.30C**).



Figs 8.29A and B: Sensory loss seen in lateral popliteal nerve/common peroneal nerve palsy.



Figs 8.30A to C: Sensory nerve supply of foot, sole with sole dermatomes.

Foot Drop

Inability to dorsiflex and evert the foot due to paralysis of the peroneal and extensor group of muscles following injury to common peroneal nerve.

Causes: Fracture neck of the fibula; leprosy; lead poisoning; iatrogenic; direct incised wound.

Clinical features: High stepping gait; loss of sensation over lateral and dorsum of the foot.

Medial Popliteal Nerve

It supplies the soleus, gastrocnemius, popliteus, plantaris, tibialis posterior, flexor digitorum longus and flexor hallucis longus. Medial popliteal nerve is rarely involved by any disease process. Trauma can cause medial popliteal nerve palsy. It is rarely involved except in open wounds.

Clinical features: Inability to plantar flex the foot; claw toes; loss of sensation in the sole of the foot (Fig. 8.31).

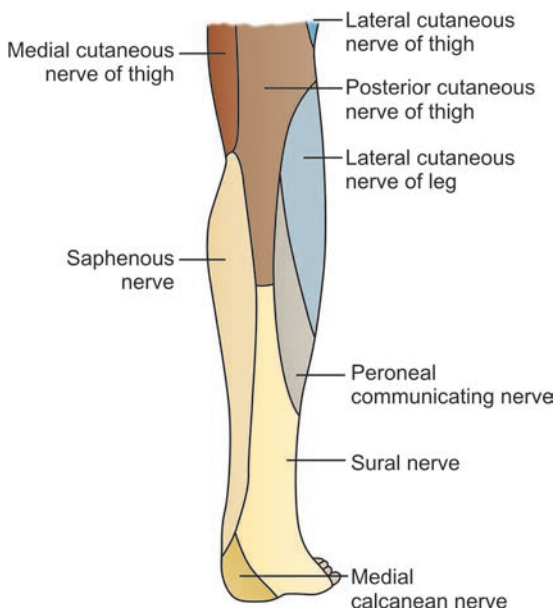


Fig. 8.31: Diagram showing sensory distribution of posterior aspect of the leg.

Axillary Nerve Injury

Axillary nerve supplies the deltoid and teres minor muscle and also sensory supply to the skin over the upper lateral aspect of the arm. Axillary nerve is affected by—Fracture neck of the humerus; dislocation of humeral head; following intramuscular injection into the deltoid. *Clinically* there will be loss of abduction of the shoulder and anaesthesia of the skin over the lateral part of the arm.

Long Thoracic Nerve Injury (Nerve of Bell)

It supplies Serratus anterior muscle. It arises from C_{5, 6, 7} cervical roots. The nerve is injured commonly in malignancy, during breast, axillary or chest wall surgeries. Often it is injured in severe brachial plexus injury. *Clinically*, when outstretched (elbow extended) arm is pushed against the wall, the inferior angle of the scapula will become prominent (*Winging of the scapula*) (Figs 8.32A and B).

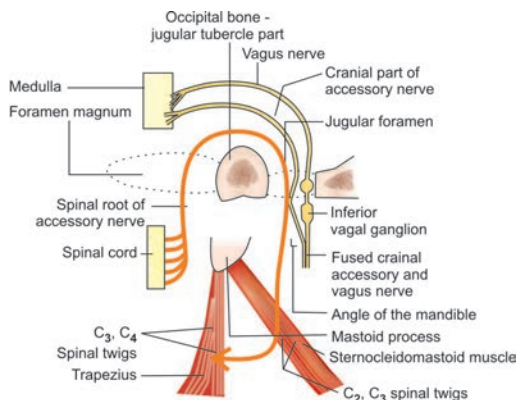
Accessory Nerve Injury

It is 11th cranial nerve having cranial and spinal roots. Cranial part begins at nucleus ambiguus and is distributed through vagal branches to muscles of palate, pharynx, and larynx. Spinal root begins from long spinal nucleus of the spinal cord between C₁ and C₅. It emerges as 5 roots from the spinal cord join to form spinal root of accessory nerve. It runs upwards to reach the foramen magnum (enters it behind the vertebral artery); joining cranial root which again gets separated. Cranial root after separation joins vagus below inferior vagal ganglion. Spinal root prior to separation from cranial root runs upwards and laterally along with 9th and 10th cranial nerves crossing jugular tubercle reaching the jugular foramen and leaving the cranium through it. Nerve descends between internal jugular vein and internal carotid artery, deep to parotid and styloid process, reaching between deep of sternomastoid muscle superficial to internal jugular vein. It is crossed by occipital artery and accompanied by sternomastoid branch of occipital artery. At the junction of upper 1/4th and lower 3/4th it pierces the anterior border of the sternomastoid muscle, emerging through the



Figs 8.32A and B: Winging of scapula is injury to long thoracic nerve of Bell—paralysing the serratus anterior muscle.

posterior border into the posterior triangle; entering the anterior margin of the trapezius 5 cm above the clavicle. It is communicated with spinal nerves on the deep surfaces of both sternomastoid and trapezius muscles through $C_{2,3}$ and $C_{3,4}$ roots. Nerve supplies sternomastoid and trapezius muscles. Nerve may be affected in advanced secondaries in neck; block dissection (radical) of the neck (**Figs 8.33A and B**). Clinically, there will be wasting of trapezius muscle; drooping of the shoulder; inability to elevate the shoulder against resistance. Sternomastoid also can



A



B

Figs 8.33A and B: Accessory nerve anatomy (11th cranial nerve). Wasting of trapezius and inability to shrug the shoulder are the typical features of accessory nerve injury.

be checked for power by turning the patient neck to opposite side against resistance.

Hypoglossal Nerve Injury

It is 12th cranial nerve arising from hypoglossal nucleus of medulla in the floor of the 4th ventricle as 10-15 roots which soon joins to form 2 bundles which later join to form single trunk. It comes out of the skull through hypoglossal canal/anterior condylar canal of occipital bone; it travels initially deep to internal jugular vein, later between internal jugular vein and

Different joints with their innervation and various muscle actions			
Shoulder	Flexion Extension Abduction Adduction	C _{5,6} C _{5,6} C _{5,6}	Nerve to pectoralis major, circumflex nerve to deltoid Thoracodorsal nerve – latissimus dorsi Deltoid muscle – axillary nerve Pectoralis major, latissimus dorsi, biceps
Elbow	Flexion Extension	C _{5,6} C _{6,7,8}	Brachialis, biceps, brachioradialis – musculocutaneous nerve Triceps, anconeus – radial nerve
Wrist	Flexion Extension Abduction Adduction	C _{6,7,8} C _{6,7,8} C _{6,7,8}	Flexor carpi radialis, flexor carpi ulnaris – median and ulnar nerves Extensor carpi radialis longus, extensor carpi radialis brevis, extensor carpi ulnaris – radial nerve Flexor carpi radialis, extensor carpi radialis longus and brevis, abductor pollicis longus and extensor pollicis brevis Flexor carpi ulnaris, extensor carpi ulnaris
Hip	Flexion Extension Abduction Adduction Medial rotation Lateral rotation	L _{2,3,4} L ₅ , S _{1,2} L _{4,5} , S ₁ L _{2,3,4}	Psoas major, iliacus – lumbar and femoral nerves Gluteus maximus and hamstrings – inferior gluteal nerve Gluteus medius and minimus – superior gluteal nerve Adductor longus, brevis, magnus – obturator nerve Tensor fascia lata, anterior fibres of gluteus medius and minimus Two obturators, two gemelli, quadratus femoris
Knee	Flexion Extension	L _{4,5} , S _{1,2} L _{2,3,4}	Biceps femoris, semitendinosus, semimembranosus – sciatic nerve Quadriceps femoris – femoral nerve
Ankle	Dorsiflexion Plantar flexion	L _{4,5} , S ₁ L ₅ , S _{1,2}	Tibialis anterior – deep peroneal nerve (anterior tibial nerve) Gastrocnemius and soleus – posterior tibial nerve (tibial nerve)
Foot	Inversion (is more than eversion) Eversion	L _{4,5} L ₅ , S ₁	Tibialis anterior and tibialis posterior Peroneus longus and peroneus brevis – superficial peroneal nerve

internal carotid artery, descending in relation to front of vagus, deep to parotid, styloid process, and posterior belly of digastric, stylohyoid, posterior auricular and occipital arteries. Near lower margin of the posterior belly of digastric, it curves anteriorly hooking lower sternomastoid branch of occipital artery; crossing internal and external carotid arteries, loop of lingual artery; running deep to posterior belly of digastric reaching submandibular salivary gland. It passes on the hyoglossus and geniohyoid deep to submandibular salivary gland and mylohyoid to reach the tongue. It is communicated by fibres from C₁ spinal nerve. It supplies all intrinsic and extrinsic muscles of the tongue except palatoglossus which is supplied by cranial part of accessory nerve through vagus. C₁ component gives meningeal branch supplying the meninges of anterior part of posterior cranial fossa. C₁ also supplies thyrohyoid and geniohyoid muscles; its descending hypoglossii branch forms upper root of ansa cervicalis. Nerve can be affected in advanced neck secondaries, malignant submandibular salivary gland, radical neck dissection and submandibular

salivary gland excision surgery. When it is involved, there will be wasting of the tongue on that side; while protruding outwards, tongue will deviate towards *same* side (**Fig. 8.34**).

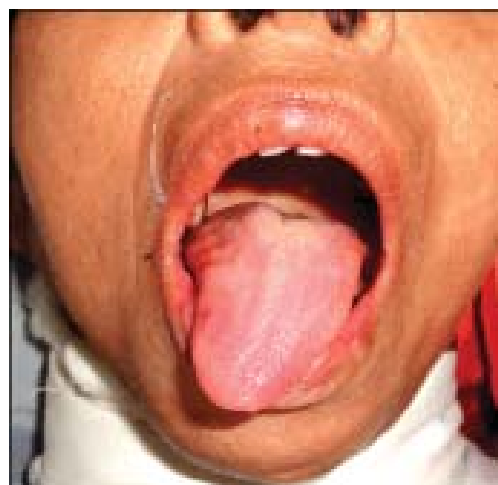


Fig. 8.34: Hypoglossal nerve palsy. Here tongue deviates towards same side. Wasting of tongue muscles on the same side is evident.

9

Examination of Muscles, Tendons and Fasciae

Sound clinical knowledge of muscle and tendon diseases is essential. It also needs a good anatomical knowledge of the muscle, its origin, insertion, actions and nerve supply.

History

Pain: Pain is the main feature in degenerative diseases of the tendons, muscles, ligaments or fasciae. Pain in epicondyles in *tennis elbow* (lateral) or *Golfer's elbow* (medial) is common. Pain of the tendon is felt in tendonitis like patellar or Achilles' tendons. In tenosynovitis, pain is felt along the tendon like de Quervain's tenosynovitis. Rest pain along the distribution of the median nerve is common in carpal tunnel syndrome.

Deformity is the symptom as well as the inspectory finding. Patients always observe the deformity and tell in the history.

General Examination

Pallor and other associates relevant features should be looked for along with nutrition, neurological examination, etc. Specific deformities are seen often in conditions like leprosy.

Local Examination (Look/Feel/ Measure/Move)

Inspection

Deformity is the commonest and specific for certain conditions. In *Dupuytren's contracture* there is flexion of ring and little fingers. *Volkman's ischaemic contracture* causes extension of wrist and metacarpophalangeal joints with flexion of interphalangeal

joints. When the wrist is fully flexed, fingers can be extended – *Volkman's sign*.

Swelling is seen in torn muscle. Tenosynovitis causes swelling of the tendon.

Skin over the area or swelling for scar/colour/oedema/sinuses/asymmetry, abnormal wrinkles, etc. should be asked for.

Wasting of the muscles should be inspected. Shape, wasting, hypertrophy, irregularity, displacement of the muscle should be looked for. Muscle should be inspected *at rest as well as with contraction*. It should be compared to other muscles and opposite side muscles.

Length of the limb, discrepancies on inspection should be observed and noted.

Palpation

Look for rise in skin temperature/presence of pitting when oedema is there.

Tenderness is elicited at the sites of the degenerative pathology whether it is epicondylitis or tendinitis.

Palpation of the swelling for all its features like any other swelling (see Chapter 3 on Swelling) should be carried out.

Measure the real and apparent lengths of the limb and compare to opposite side.

Muscle should be palpated at rest as well as with contraction. When muscle is at rest intramuscular swelling moves in right angle to the length of the muscle; when muscle is contracted intramuscular lump becomes immobile. Ruptured muscle when felt will

be tender often with a swelling. There will be a depression when relaxed; but a firm swelling over the edge of the ruptured muscle with a sharp depression at the site of tear is typical on contraction. Haematoma may be felt in a ruptured muscle.

Movements of the joints: Active movement is the one which patient does and shows to the examiner; whose range, abnormal mobility or restrictions should be observed. Passive movement is the one which examiner elicits using his hands.

Neurological Examination

Neurological examination should be done like in carpal tunnel syndrome for median nerve palsy. Motor power and reflexes should be checked.

Peripheral pulses should be examined for blood supply.

Relevant Systemic Examination

Relevant systemic examination is a must.

Carpal Tunnel Syndrome

It is the classical example of stenosing tenosynovitis. Refer for detail Chapter 8, pg 212.

Stenosing Tenosynovitis/Tenovaginitis

Trigger finger: It is of unknown aetiology wherein extension of the affected finger is difficult; finger gets 'locked' fully in flexed position; flexed finger can be extended with a 'sudden click' like a trigger of a pistol using excessive effort often with other hand. It is common in middle aged women. There will be a thickened nodule in the long flexor tendon sheath adjacent to head of the metacarpal or constricting band/ring in the synovial sheath. It is common in middle aged women. It is not painful even on clicking. Finger gets stuck in flexed position and unable to extend. It is commonly seen in middle or ring finger. Finger looks normal. Sensation is normal. It is not associated with any systemic musculoskeletal diseases. **Trigger/snapping thumb:** It is rare; seen in neonates and infants; affecting thumb with a similar snap. An orange pip like swelling may be felt over the head of the first metacarpal bone. **de Quervain's stenosing tenosynovitis:** It involves the common tendon sheath of the abductor pollicis longus and extensor pollicis brevis.

Bulge may be seen or felt over the radial styloid process or anatomical snuff box. There is difficulty in abducting and extending the thumb. Adduction of thumb is painful.

Congenital Contracture of Little Finger

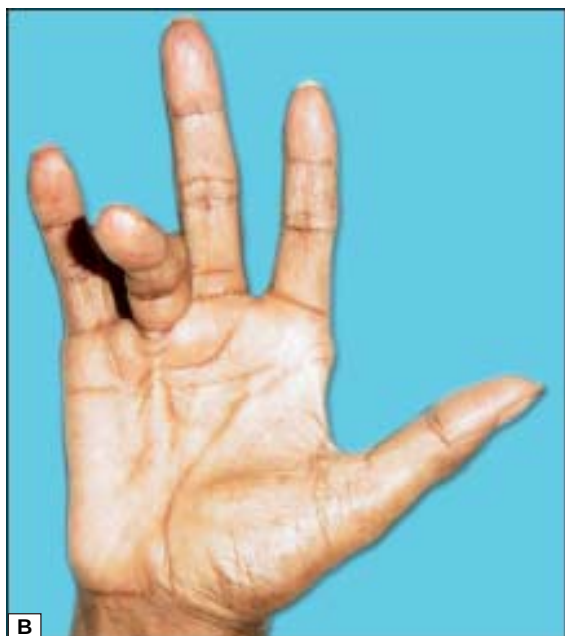
It is commonly a bilateral condition; seen in childhood; with contracture of soft tissues; mimics Dupuytren's contracture but palmar aponeurosis is normal and ring finger is not involved. Here first phalanx is hyper-extended, middle and terminal phalanges are flexed. Straightening of the finger may not be possible.

Dupuytren's Contracture

It is localised thickening of palmar aponeurosis with fibrous *nodule* formation causing flexion of ring and then little fingers. Terminal phalanx is not affected as palmar aponeurosis does not extend into terminal phalanx. It begins at and mainly involves medial aspect of the palmar aponeurosis. Eventually all fingers may get involved; joints also may be involved causing arthritis and stiff joints. Skin gets adherent to thickened palmar aponeurosis. It is often familial and bilateral (45%). It is common in males (10 times). Pain is not common; but stiffness is usual. Taut fibrous strands are seen and felt especially along the line of ring and little fingers. By flexing the wrist flexion deformity *will not get reduced*. *Garrod's pads* of fat develop over the knuckles of proximal interphalangeal joints. Dupuytren's contracture is often associated with – plantar fasciitis (5%; *Lederhose's* disease); mediastinal and retroperitoneal fibrosis; *Peyronie's* disease of penis (3%); nodules in the face and ear; *Pellegrini Steida's* disease (Myositis ossificans with calcification of commonly superior part of medial collateral ligament of knee joint). *Galezia triad* is Dupuytren's contracture; retroperitoneal fibrosis; *Peyronie's* diseases of penis. Dupuytren's contracture may be due to repeated minor trauma, cirrhosis, alcoholism, epileptics on phenytoin therapy, diabetes mellitus and other metabolic conditions. It can be often familial – autosomal dominant. Condition causes restriction of hand function and arthritis of joints (**Figs 9.1A and B**).

Burns Contracture of the Finger

It causes permanent contracture of fingers and wrist. History of burn injury will be present (**Fig. 9.2**).



Figs 9.1A and B: Dupuytren's contracture.

Mallet Finger/Base Ball Finger

It is fixed flexion deformity of terminal phalanx/distal interphalangeal joint due to rupture of extensor tendon of distal phalanx or avulsion fracture of the base of the distal phalanx near its insertion. Injury by a hard ball/object into the flexed finger tip is the cause. There is inability to extend the tip of the finger; distal phalanx of affected finger is 20° flexed; distal IP joint can be flexed to 90° and when extended it comes upto 20° flexed position; it cannot be extended further and



Fig. 9.2: Finger deformity after burns contracture.

straightened. It is serious handicap only to whom who are in need of fine movements with fingers like musicians, tailors, surgeons.

Rupture of Extensor Pollicis Longus

It is due to attrition of extensor pollicis longus tendon causing sudden rupture with a snap while working and later thumb becomes adducted with inability to extend the terminal phalanx of the thumb. It is common in females; seen in rheumatoid arthritis or as a complication of Colles' fracture.

Rheumatoid Arthritis of the Hand

Hands are commonly involved in rheumatoid arthritis. Hypertrophy of the *synovial* membrane of the joints is the initial feature. Overlying skin becomes shiny and atrophic. Wasting of muscles with swollen, spindle shaped/fusiform joints and later development of deformities occurs. Metacarpophalangeal joint is first to get affected; later proximal interphalangeal joint. Deformities are – deviation of finger towards ulnar side at metacarpophalangeal joint (varus deformity of 45° – 60°) – *ulnar drift*; fixed flexion deformity of wrist with ulnar deviation; fibrotic contraction of interossei and lumbricals causing hyperextension of the proximal interphalangeal joint and flexion of distal interphalangeal joint – *swan neck deformity*; flexion of proximal interphalangeal joint and hyperextension of distal interphalangeal joint due to attrition of middle slip of the extensor tendon – '*boutonniere*' deformity; rupture of extensor tendons at wrist level causing dropped finger (**Figs 9.3 and 9.4**).



Fig. 9.3: Button hole/Boutonniere deformity. There is flexion of proximal interphalangeal joint and hyperextension of distal interphalangeal joint.



Fig. 9.4: Swan neck deformity. It is hyperextension of proximal interphalangeal joint and flexion of distal interphalangeal joint.

Heberden's Nodes

They are bony swellings close to distal interphalangeal joints both in palmar and dorsal aspects. Dorsal aspect is more common site. They are nonspecific even though associated commonly with osteoarthritis. They are common in females. In males when it occurs it is usually solitary may be of traumatic origin by sports like base ball/cricket ball injury. All fingers may be involved but not thumb. Index finger is most commonly affected. They are nonmobile bone swellings. Radial deviation of distal phalanx with osteoarthritis of distal interphalangeal joint is usual. Small adventitious bursae may develop between swelling and skin. Often swelling over proximal interphalangeal joint may also be involved.

Ruptured Muscle Fibres

Unusual sudden excessive contraction of a normal muscle or normal contraction of a degenerated muscle can cause rupture of a muscle. Muscle rupture is common in young athletes or in old people who does unusual strenuous work. Commonly affected muscles are biceps brachii, quadriceps femoris. Pain, bruising, swelling may appear after tear. But usually tear may be unnoticed but later presents as muscle weakness, *swelling*, and limp. In relaxed state, there will be a depression at the site of the tear; when contracted swelling will be seen or felt at the edge of the depression which is firm and without any independent mobility. Swelling is free edge of the contracted partially torn muscle fibres. In complete tear, movement of muscle will be absent. All adjacent joints, arterial and nervous system should be examined. Chronic degenerative or musculoskeletal diseases should be looked for. *Rupture of long head of biceps brachii*: It occurs in bicipital groove, usually spontaneous due to a degenerative disease in elderly; presenting as either sudden pain in upper arm or more commonly swelling due to bunching of muscle when elbow is flexed. Rupture of the supraspinatus tendon occurs in elderly as a degenerative disease; presenting as sudden pain and absence of initiation of abduction. Initiation is done by body by leaning towards the affected side. Later part of abduction of the shoulder is normal. *Rupture of the Achilles' tendon*: It is seen in athletes like foot ball players where violent undue contraction of the calf muscle occurs. There may be arterial insufficiency of tendon also. Swelling is seen or felt in the posterior lower part of leg while plantar flexion of the ankle is typical.

Intramuscular Haematoma

Here injury is direct with a tear in the muscle which also injures the intramuscular blood vessels. It is often seen in people who take *anticoagulants* and who are suffering from blood dyscrasias. Pain is very common which is present at rest and aggravated by muscle movements. Firm, tender well localised swelling in the muscle is felt even in relaxed state. Tenderness in the swelling disappears in few days but pain will persist for few weeks while contracting the muscle. This firm/*hard* swelling is longitudinally ovoid and parallel to muscle fibres. Usually swelling is hard but occasionally liquefaction of haematoma makes it soft

and fluctuant. On contracting the muscle swelling which was well felt may become indistinct. Gastrocnemius is the commonest muscle affected.

Muscle Hernia

It is bulging out of the muscle especially during contraction through a defect in its fibrous sheath covering. This is obvious and significant only when it occurs in muscles having thick fibrous sheath like anterior compartment of leg, lateral abdominal and back muscles. Size of hernia changes and depends on the amount of contraction developed in the muscle. When muscle is relaxed swelling is absent but a distinct defect is palpable in the fascia. To diagnose muscle hernia these two features are essential, i.e.— while contracting a bulge felt through the defect; on relaxing the distinct fascial defect is felt. Muscle hernia is common in lumbar region and calf.

Intra- or Intermuscular Lipoma

Lipoma can occur within the muscle or in between muscles. Such lipoma may interfere with function of the muscle. Swelling in the muscle which is smooth, soft or firm with lobulations may be felt but when contracted swelling often may become indistinct, immobile and hard. Occasionally such lipoma which was not palpable becomes suddenly palpable during exercise due to its bursting out through muscle fibres making it painful palpable swelling. Intra- or intermuscular lipoma is common in back of the trunk as fat content of these muscles are more than muscles in other part of the body. Initially such lipoma is not palpable and grows silently to attain large size. Usually such lipomas are single.

Myositis Ossificans

It is calcification and eventual ossification of part of the injured muscle which is usually associated with fracture of adjacent bone. It is seen in lower part of the brachialis muscle after supracondylar fracture of the humerus or quadriceps femoris in fracture femur. *Features are:* Inability to use the muscle; stiff adjacent joint; painful forced movements. Ossified part attains the involved muscle shape and is fixed to the underlying bone. This ossified muscle is continuous with callus underneath. Often this ossified muscle may be mistaken

for callus underneath. Muscle over a callus will function normally; whereas ossified muscle is functionless. Features of old fracture will be there with restricted joint movements.

Tennis Elbow

It is *lateral epicondylitis* of the humerus presenting as pain in lateral epicondyle of the humerus at the attachment of common extensor forearm muscles. When wrist is extended against resistance pain is aggravated. There is localised tenderness at the lateral epicondyle of the humerus (Refer Chapter 30, pg 685).

Golfer's Elbow

It is pain and localised tenderness over the *medial epicondyle* adjacent to origin of common flexor tendon of the forearm which is aggravated by flexing the wrist against resistance (Refer Chapter 30, pg 685).

Plantar Fasciitis

Tear or bony spur at the attachment of the plantar fascia to the calcaneum causes unbearable pain in the heel while walking.

Supraspinatus Tendinitis

Supraspinatus muscle from its origin from supraspinous fossa of the scapula is inserted to upper impression of the greater tubercle of the head of the humerus. Degenerative process in this tendon often with calcification is seen in middle aged or elderly males causing pain in the shoulder during middle third of the abduction and external rotation of the shoulder, which occurs because when the head of the humerus comes in contact with the acromion causes compression of the degenerated supraspinatus tendon. First 60° of abduction is painless; next 60° abduction is painful; further abduction after 120° is painless causing typical *painful arc syndrome*. Stiffness of shoulder develops causing frozen shoulder. Pain gradually subsides while stiffness increases; later stiffness persists but pain subsides in 3 months; in further 3 months stiffness also slowly subsides. Spontaneous rupture of the degenerated supraspinatus tendon can occur. Calcification in the tendon can be confirmed by X-ray.

Volkmann's Ischaemic Contracture

It is development of muscular infarction initially acute later chronic causing subsequent contracture. There is shortening of the long flexors of the forearm due to ischaemic fibrosis of the muscle (aseptic muscle necrosis and fibrosis). *Causes:* Supracondylar fracture (commonest) injuring brachial artery which bleeds or undergoes spasm causing raised pressure in the compartment which again further compromises the blood supply of the muscle; a tight plaster which compresses the artery blocking the blood flow; arterial embolism. Burns, closed forearm crush injury, intravenous chemotherapy are other causes. *Features in acute phase:* Condition is common in young individual; history of trauma is evident; pain in the forearm and fingers (under the plaster is typical); loss of finger movements (mainly finger extension); cold skin; paraesthesia (due to ischaemia of median and anterior interosseous nerves) severe burning pain or pins and needles sensation due to ischaemic neuritis; absence radial pulse; pallor; oedema of the forearm (puffiness). *Chronic phase:* Once acute phase subsides gradually, pain disappears but deformity persists with inability to extend fingers. By flexing the wrist fingers can be



Fig. 9.5: Volkmann's sign in Volkmann's ischaemic contracture. When wrist is flexed fully fingers can be extended at interphalangeal joints. It is not possible in Dupuytren's contracture.

extended – *Volkmann's sign*. There is claw hand deformity. Fingers are in acutely flexed position. Forceful passive finger extension is uncomfortable and often painful. Metacarpophalangeal joint is extended (**Fig. 9.5**).

10

Examination of Oral Cavity

Oral cavity is a wide area which includes lips, vestibule, gums, teeth, cheeks, tongue, palate, and floor of the mouth. Vestibule is a smaller outer portion bounded externally by lips and cheeks; internally by teeth and gums. Parotid duct opens into the cheek opposite the crown of upper 2nd molar tooth. Numerous mucus glands that are situated in the submucosa of lips and cheeks open into the vestibule.

History taking begins with:

Name:

Age:

Sex:

Occupation:

Address:

Agriculturists who are constantly exposed to sunlight are prone to develop carcinoma lip—*countryman's lip*. Carcinoma oral cavity is more common in males. Cleft lip and palate is seen in newborns. Mucus cysts can occur at any age group. Australian Caucasians commonly develop lip cancers. It is less common in Negroes.

History

History of Present Illness

History of swelling: Mucus cyst of lip or cheek presents as painless swelling of long duration. It is painless. Duration, progress, presence of pain should be asked for. Carcinoma often can present as swelling of short duration. Lip cancer is slowly progressive and so may have long duration. Carcinoma of cheek and tongue is rapidly progressive and is having short duration. Minor salivary tumour in palate and lip presents as swelling.

History of ulcer: Ulcer in the oral cavity is common. It can be aphthous ulcer/syphilitic ulcer/traumatic

ulcer/tuberculous ulcer/malignant ulcer. Aphthous ulcer is painful. Malignant ulcer is painless to begin with but becomes painful once it infiltrates or gets infected. Origin of ulcer, duration, progress should be asked for.

History of pain: Site of pain, radiation, referred pain, severity of pain, pain over the adjacent mandible, whether pain restricts mouth opening or swallowing should be asked for. Pain may radiate or get referred to ear through lingual nerve or inferior alveolar nerve (through auriculotemporal branch of mandibular nerve). Dental ulcer on the margin of the tongue is painful. Aphthous ulcer is painful. Tuberculous ulcer may not have any pain. Retention cyst, leukoplakia, early oral cancers are painless. In late cases of carcinoma pain develops due to deeper infiltration (to nerves), and sepsis.

Excessive salivation is common in oral cancers especially in carcinoma tongue.

Inability to protrude the tongue out is common in carcinoma tongue involving floor of the mouth or infiltrating the genioglossus muscle.

Difficulty in speech is common in carcinoma tongue. It is also often seen in painful aphthous ulcers.

Voice change or dysphagia may be the feature of carcinoma posterior third of tongue.

Dentition, recent history of loosening of teeth, falling of teeth is important as it may be due to underlying carcinoma.

Bleeding, halitosis (foul smelling breath), *altered taste sensation*, cough, haemoptysis are other history to be asked. History of fever suggests infection, bronchopneumonia (due to aspiration especially in carcinoma tongue).

Symptoms suggestive of local invasion (difficulty in opening the mouth, mandibular pain, loss of sensation in the chin or gums); cervical lymph nodal spread (swelling in the neck, duration, pain, ulceration).

Past History

Past history of oral ulcers, treatment received in the form of surgery, radiotherapy, chemotherapy has to be noted. History suggestive of leukoplakia also should be asked.

Personal History

Smoking, alcohol intake, spicy food, pan chewing (betel nut, supari, Khaini, etc.) are important causes for carcinoma. It is also important to note how long patient keeps the pan in the cheek which will increase the irritation. *Reverse smoking* is often related to carcinoma hard palate.

General Examination

Detailed general examination is very essential. Anaemia/oedema/jaundice/clubbing/lymphadenopathy/radial pulse/blood pressure/raise in temperature/attitude of the patient/nutritional assessment by skin texture, subcutaneous fat, weight, body mass index/any other relevant findings should be mentioned. Cachexia signifies advanced malignancy. Halitosis may be found. Temperature may be raised.

Local Examination

Inspection

Inspection of the oral cavity should be done using proper and adequate light. A *spatula* should be used eventually to inspect posterior aspect of the oral cavity. Inspection is done in order – lips, cheeks, teeth, gums, tongue, floor of the mouth, palate, tonsils, posterior aspect (**Figs 10.1A to D**).

Inspection of the Lip

Lips are two fleshy folds lined by skin outside and mucous membrane inside. Upper lip is bounded by nose and nasolabial groove. Lower lip is bounded by cheek and labiomental groove. Orbicularis oris forms the muscular bulk of the lip which encircles the lip and is supplied by facial nerve. *Frenulum* in the midline

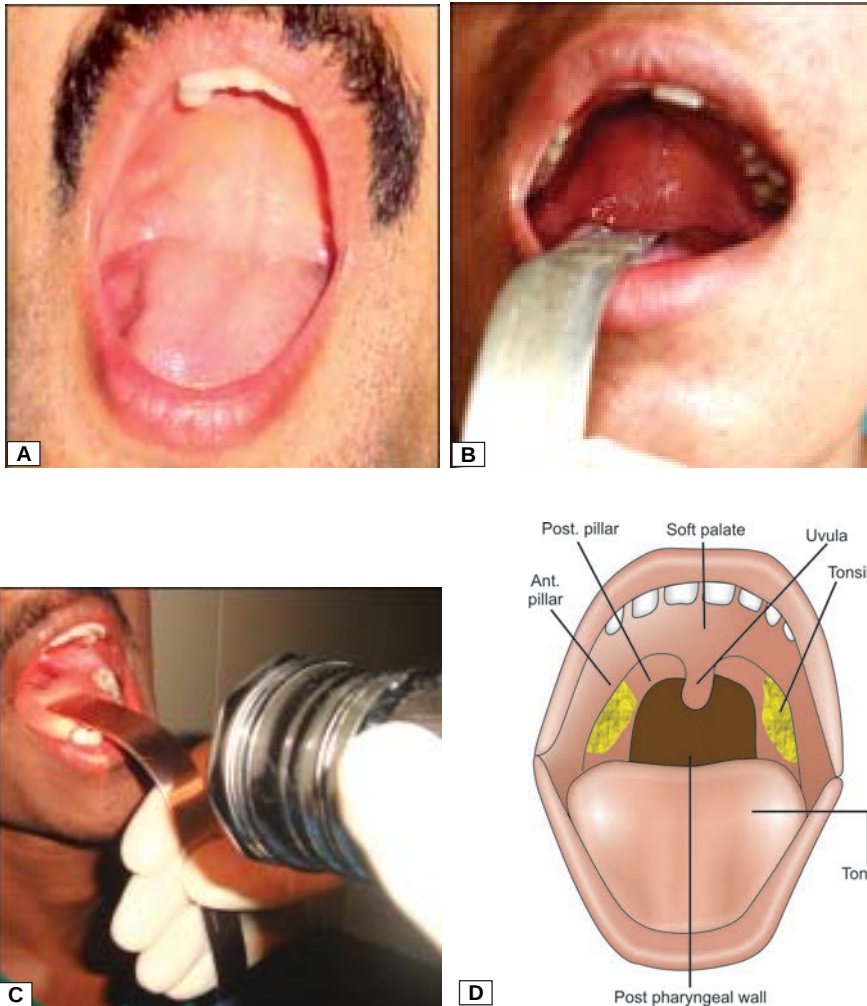
in upper and lower lips joins lip to the gums. *Vermilion border* is red border of the lip where skin part merges gradually into the mucous membrane part. It contains wet line inside and a dry line outside. Small rounded nodule at the centre of the lowest part of the upper lip is called as *tubercle*. A depression running from tubercle to nostrils is called as *philtrum*. The corner where upper and lower lips meet at right and left angles are called as *commissures* of lip. 5 mm elevation of mucous membrane posterior to commissure is called as *commissural papule*. Upper lip drains into upper deep cervical nodes. Centre of lower lip drains into *submental nodes* then to upper deep cervical nodes. Lateral part of lower lip drains into submandibular lymph nodes then to *middle cervical nodes*. Lymph from angles of mouth drains into both nodes of upper and lower lips. Lips are red or reddish brown in young. It is often brownish in smokers.

Cleft lip and *cleft palate* are obvious. Its type, side, extent should be noted down. *Macrocheilia* is enlarged lip which is common in upper lip may be due to lymphangioma or haemangioma (soft, bluish with compressibility and emptying is typical) (**Figs 10.2 and 10.3**).

Blackish pigmentation can occur in lip or cheek in Addison's disease. *Bluish* pigment spots are seen in lower lip, cheek or palate in Peutz-Jegher's syndrome along with multiple polyps in the small bowel, occasionally in colon inherited as autosomal dominant familial disease.

Acute ulcers like *aphthous ulcers* can occur in lip. *Aphthous ulcers* (aphthous – Greek – to set on fire; in USA called as 'Canker sore') are common in younger age group; self limiting in 7-14 days; related to stress or nutritional deficiency; are small, often multiple superficial painful erosive lesions with whitish floor with yellowish and hyperaemic margin. In cold weather lips may get *cracked* mainly in the midline (**Fig. 10.4**).

Carcinoma lip is common in lower lip. It is slow growing tumour initially may present as a proliferative/nodular or ulcerative lesion with whitish plaques/red areas/necrotic tissues over the surface (**Fig. 10.5**). This lesion should be inspected in detail for margin (regular/irregular/well defined/ill defined); edge (everted/raised); floor; surrounding area; angles of the lip. Often there will be oedema surrounding the lesion. Entire



Figs 10.1A to D: Proper inspection of the oral cavity is essential part of the examination. Often spatula/tongue depressor and a good light source should be used to inspect the oral cavity.

lip may get enlarged. *Discharge* on the surface may be serous/serosanguinous/purulent.

Minor salivary gland tumour can occur in upper lip. It usually begins as a swelling in the upper lip; slowly progressing eventually forming an ulcer over the summit of the swelling. *Primary syphilitic chancre* in the lip (common in upper lip) is pink painless macule to begin with; becomes papule and later superficial ulcer with thick crust on the floor and often ulcer may be painful. These ulcers eventually heal with a permanent fine superficial scar. Angular stomatitis (cheilosis), syphilitic *rhagades* (secondary syphilis),

vitamin deficiency (riboflavin) ulcers, denture induced, cracks due to allergy to dentures/lipsticks can develop in the angles of the mouth (commissures). *Perleche* (French – to lick) is angular stomatitis seen in children as a simpler infection which does not extend to mucus surface and heals without scarring. Stomatitis in syphilis extends to mucous membrane and heals with a scar.

Carbuncle of upper lip even though now rare; can be dangerous due to development of fatal cavernous sinus thrombosis due to spread of sepsis through dangerous zone. It may cause thrombophlebitis of



Fig. 10.2: Cleft lip and palate in a child.



Fig. 10.3: Haemangioma of upper lip.



Fig. 10.4: Aphthous ulcer over the lip and tongue.



Fig. 10.5: Carcinoma lip—proliferative lesion.

ophthalmic plexus of veins leading into upper eyelid oedema. *Actinic cheilitis* is common in lower lip; is due to exposure to sun light; present as recurring small blisters with epithelial exfoliation; recurrent lesions are premalignant. *Keratoacanthoma* (molluscum sebaceum) can occur in lower lip which is entirely benign but mimics carcinoma. Retention *mucus* cyst is common in lower lip which is blue, well localised; smooth (fluctuant and transilluminating).

Inspection of the Cheek

It is large fleshy flap one on each side covering the vestibule. It contains skin, superficial fascia with facial

muscles, parotid duct, mucus glands, buccinator with buccopharyngeal fascia, submucosa and mucous membrane. Buccal pad of fat lies on the buccinator partly deep and partly in front of masseter.

Cheek is inspected for leukoplakia, mucous cyst, swellings, papilloma, and carcinoma (**Fig. 10.6**). Pigmentation similar to lips can also develop in cheeks. *Leukoplakia* is whitish patch in the mucosa of the oral cavity that cannot be characterised clinically or pathologically to any other disease. It is a premalignant disease. It is common in smokers and who chew pan (20%). It has got 4% chances of turning into malignancy.



Fig. 10.6: Cheek inspected using a spatula/depressor.



Fig. 10.7: Severe mucositis mainly in cheek.

Erythroplakia, submucosal fibrosis are other conditions to be looked for in cheek (**Figs 10.7 and 10.8**).

Carcinoma cheek needs special mention. Cheek is common site of carcinoma in oral cavity. It can be either ulcerative or proliferative lesion. Margin, size, shape, edge, floor of the lesion and surrounding area should be inspected. Once carcinoma infiltrates deep into the pterygoid muscle, *trismus* develops.

Trismus is inability to open the mouth adequately. Trismus is decreased interincisor distance between upper and lower jaws (**Figs 10.9A to 10.10B**).



Fig. 10.8: Carcinoma lip and cheek.



Figs 10.9A and B: Carcinoma cheek in two different patients. Tongue depressor/spatula use is helpful in proper inspection of the cheek.



Figs 10.10A and B: Trismus in carcinoma cheek suggests involvement of pterygoids and soft tissue. It is checked by placing fingers (of patient ideally) perpendicularly between opened jaws.

Grading of trismus: Interincisor distance more than 3.5 cm is – normal. Grade I is between 3.0–3.5 cm. Grade II is between 2.0–3 cm. Grade III is less than 2 cm.

Inspection of Teeth and Gums

Teeth should be counted. Primary dentition is 20 in children. Secondary permanent dentition is 32 in adult. 2 incisors; 1 canine; 2 premolars; 3 molars (2123). Third molar tooth (total four) are last to erupt on each sides at late teenage. One or more tooth may be absent; changed spacing; deformities are common. Teeth may be green in infants with jaundice; tetracycline given in early childhood may stain the teeth. Excess fluorides in drinking water may cause black pits in the teeth. Transverse ridge with curved notching is seen in rickets.

Tartar (precipitated calcium in saliva) deposition occurs on lingual sides of lower incisors due to constant exposure to calcium rich saliva from submandibular salivary gland. Tartar may precipitate pyorrhoea alveolaris (**Fig. 10.11**).



Fig. 10.11: Oral cavity inspection always includes inspection of dentition properly.

Tooth which is prevented from erupting by other teeth is called as *impacted tooth*. Mandibular 3rd molar is commonly impacted tooth (*wisdom tooth*). *Incompletely erupted* mandibular 3rd molar commonly suppurates and is dangerous, and is *common cause of trismus*. *Dead tooth* is less white or bluish gray and insensitive to ice placed over it. *Hutchinson's teeth* are seen in congenital syphilis; only secondary dentitions are affected; common in upper central incisor; small notched incisor is typical. *Screwdriver tooth* is also seen in congenital syphilis. *Moon's molar* is dome shaped first molar – seen in congenital syphilis.

Gingivae or gums are mucous membrane covering the alveolar process of the jaws. It is pink in colour in healthy person. It is spotted with brown melanin pigment in dark skin people and people from Mediterranean region. It is pigmented in smokers, pan chewers. Gingival margin is occlusal border at which gingiva meets teeth. *Free gingiva* is gingival part encircling the tooth forming a gingival sulcus. *Attached gingiva* is mucosa which is firmly bound to the underlying bone. *Alveolar mucosa* is movable vascular mucosa and less attached to bone. Lips should be



Fig. 10.12: Gingivitis with pigmentation and ulcer.

everted properly to inspect the gums. Proper light is needed. Gums recede as age advances (**Fig. 10.12**).

Vincent's gingivitis/stomatitis (Trench mouth) is an inflammatory condition with ulcer and *pseudomembrane* in the gums and adjoining mucous membrane. It is due to *Borrelia vincentii* and fusiform fusiformis bacteria. Purple red lesion may be evident in gums in *cancrum oris*. It is commonly observed in molar or premolar region. Condition has got foul smell. *Cancrum oris (Noma)* is an infective gangrene, rapidly progressing into the bone and soft tissues in cheek with destruction (*Phagedena*). It is common in children after measles, gastroenteritis, typhoid, and bronchopneumonia.

Swollen gum is seen in dental abscess. Swelling in the gums which is localised is called as *epulis*. *Blue line* in gums is observed in those who work in lead industries. They are better observed using magnifying lens. Similar bismuth or mercury lines are also seen.

Swollen, livid, spongy, tender bleeding gums with loose teeth are seen in *scurvy*. *Generalised hyperplastic progressive gingivitis* is seen in children often after antiepileptic drugs. *Hyperplastic gums* are also seen in children with acute leukaemia due to immature granulocytes and secondary infection. Gums bleed on touch and there is fever.

Inspection of the Tongue

Tongue is a muscular, glandular, vascular flat organ. Anterior 2/3rd is termed as body; posterior 1/3rd is

base/root. Superior surface is *dorsum of the tongue*. It is an essential organ of taste. Tongue is important in speech, mastication and swallowing. *Filiform papillae* are located in anterior 2/3rd of the dorsum of tongue and are numerous, fine, hair like. *Fungiform papillae* are mushroom shaped, deep red, larger, sparsely located near the tip of the tongue. Large, red, leaf like *foliate papillae* are located in posterior third of tongue on lateral aspect which contains taste buds. *Circumvallate papillae* are 8-12 in number mushroom shaped, arranged in large V shaped row near posterior third of the dorsum tongue and contains plenty of taste buds. Small circular opening just posterior to this V row in the midline is called as *foramen caecum* which is the remnant of thyroglossal duct. Shallow groove just behind the circumvallate papilla on either sides of the foramen caecum is called as *terminal sulcus*. Numerous mucin glands and lymph follicles in the posterior third of the dorsum of tongue is called as *lingual tonsil*. *Posterior third of the tongue is difficult to inspect*; it needs headlight, and spatula. It is better felt than seen. *Ventral surface* is smooth, has a median fold, *frenulum linguae* and deep lingual veins on either side. *Lingual frenulum* is attached about 10-15 mm below the mandibular central incisor tooth. *In tongue tie* it is only 3-4 mm below the central incisor. It is congenital short frenulum; which is better seen when tip of the tongue is rolled upwards. Child may not protrude the tongue and there may be speech difficulties. Tongue is examined properly often by wrapping it with a damp gauze and pulling it out. Its anterior surface, dorsum, ventral surface, margins should be inspected (**Figs 10.13 and 10.55**).

Macroglossia (Megaloglossia/pachyglossia) is a disorder in which the tongue is larger than normal. Macroglossia is usually caused by an increase in the amount (volume) of tissue on the tongue, rather than by a growth, such as a tumour. It is often seen in haemangioma, lymphangioma, muscular macroglossia (in cretins), acromegaly, *Beckwith-Wiedemann syndrome* (hypoglycaemia, abdominal wall defects, Wilm's tumour, macroglossia, adrenal tumour), Down's syndrome, mucopolysaccharidoses, primary amyloidosis, occasionally plexiform neurofibromatosis. Often it causes functional and cosmetic problems (**Fig. 10.14**).

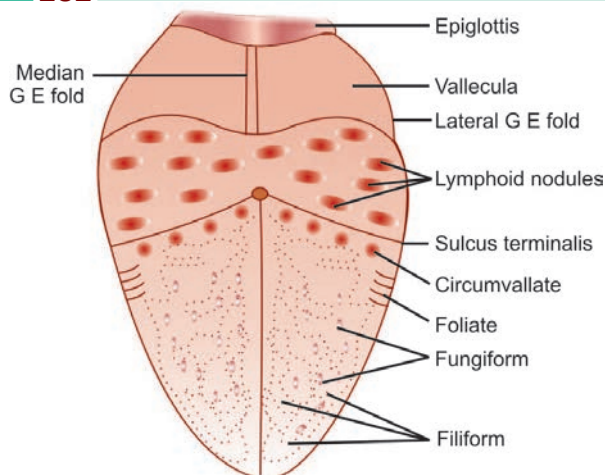


Fig. 10.13: Anatomy of tongue.



Fig. 10.14: Macroglossia in a Down's syndrome patient.

Chronic *superficial glossitis*, leukoplakic patches, mucous membrane hyperkeratosis causing *black hairy tongue* (filiform papillary hypertrophy), discoloured tongue in *Aspergillus* fungus infection, fissure, ulcers, swellings, etc. should be inspected. Congenital fissure is *transverse*. It appears at the age of 3 years and persists later for life. Syphilitic fissure is *longitudinal* with *denuded* intervening epithelium. Carcinoma can present as a fissure. *Median rhomboid glossitis* is a rhomboid mass in the midline posteriorly in front of the foramen caecum of tongue; probably due to persistent tuberculum impar; extends deep into the tongue muscles; with well defined margin; without any papillae; with slight induration on it mimicking

carcinoma. *Glossitis migrans/geographical tongue* can be idiopathic in children or secondary to major surgery or peritonitis causing bright red colour with yellowish white margin. Its location and pattern changes within 2 days. If it is of idiopathic origin it subsides in 7 days; but in secondary type it subsides only once patient recovers from main disease (Figs 10.15 and 10.16).



Fig. 10.15: Severe glossitis.

Ulcer in tongue when present, its size, location, margin, edge, extension and surrounding area should be inspected. Inability to protrude the tongue is called as *ankyloglossia*. It is seen in carcinoma tongue infiltrating the floor of the mouth. Tongue may deviate towards same side (with wasting tongue muscle on the same side) if there is *hypoglossal nerve palsy* due to nodal infiltration or carcinoma tongue infiltrating the nerve (Figs 10.17 to 10.19).

Leukoplakia (Greek-white plate) in tongue is typical lesion. Early lesion is thin, crinkled and pearly. Late lesions are large, creamy white, thick often desquamated with beefy red colour. Sir Henry Butlin said, 'tongue looks as though it had been covered with white paint that had hardened, dried and cracked'. Early cases are better inspected by pressing a glass slide on the surface.

Papilloma, neurofibroma can occur in the tongue. Size, shape, surface, margin should be mentioned. Aphthous/dental ulcers are common on the *lateral margin*. Tuberculous ulcer is common in *tip* of the

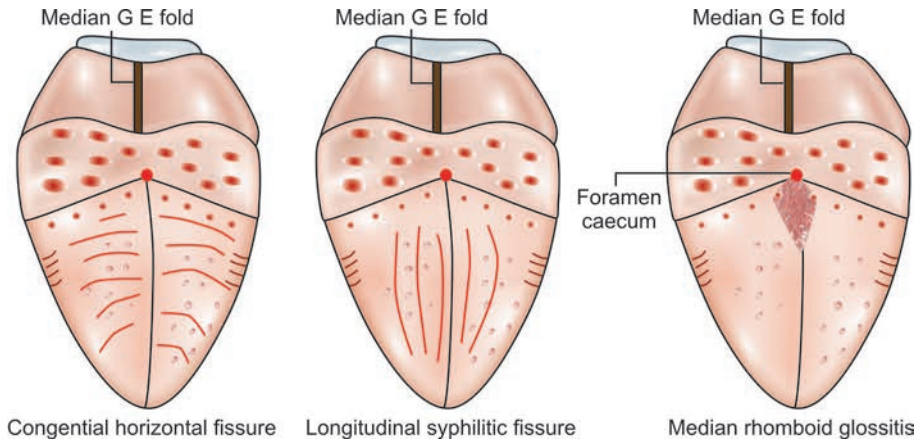


Fig. 10.16: Congenital fissures of tongue are transverse; syphilitic fissures are longitudinal. Median rhomboid glossitis is persistent tuberculum impar.

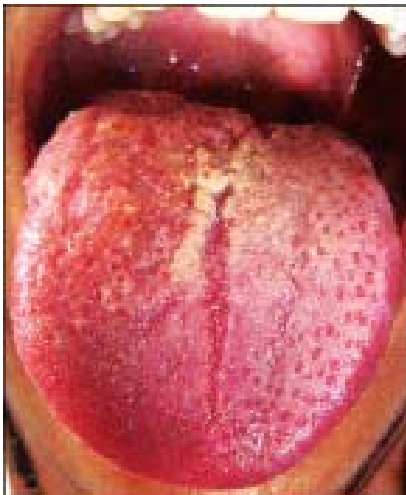


Fig. 10.17: Tongue fissure. It could be a presentation of carcinoma.

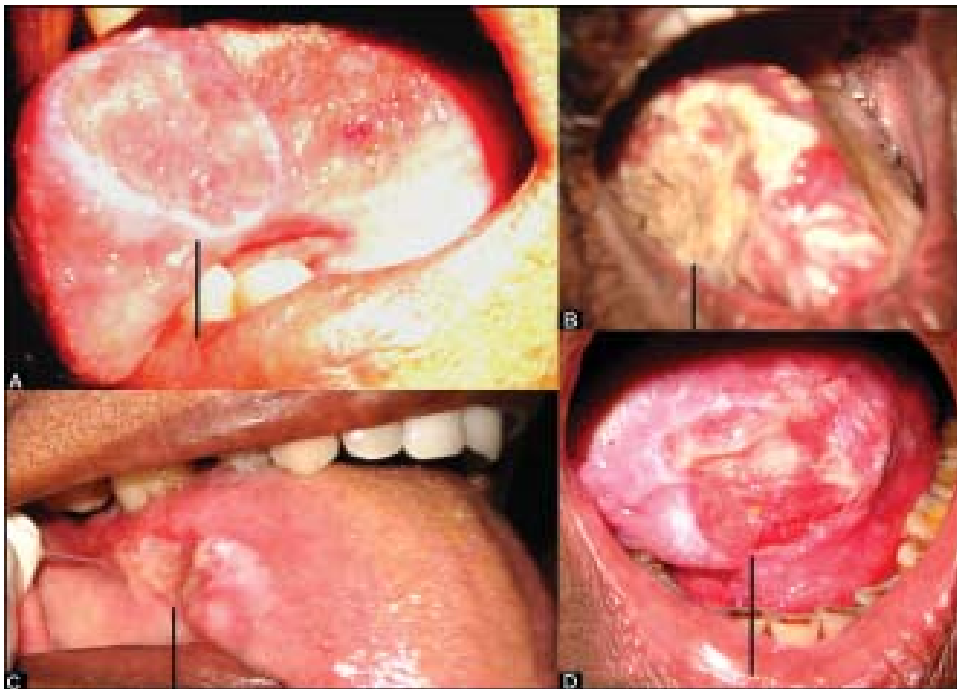
tongue. Syphilitic gummatous ulcer is common on *dorsum of tongue*. Carcinoma is common in *margin*. Lingual thyroid may be the only thyroid existing in the region of *foramen caecum* as a smooth swelling. *Tongue tremor* is checked with tongue inside the oral cavity (in protruded tongue fasciculation may mimic the tremor).

Lichen planus in tongue are delicate bluish white silver nitrate coloured lesion; often difficult to differentiate from carcinoma; but there are also lesions over the front of wrists and shin.

Inspection of the Floor of the Mouth

It is U shaped area bounded by lower gum and oral tongue. It ends posteriorly at the insertion of anterior tonsillar pillar into the tongue. Sublingual papilla is present on each side of the frenulum; on summit of which is the opening of the duct (Wharton's) of submandibular salivary gland. Laterally and behind this papilla, sublingual fold is present which overlies the sublingual gland. Genioglossus and geniohyoid muscles are deeper to it. On either side mylohyoid muscles forms the muscular part of the floor of the mouth. It arises from mylohyoid ridge of the mandible extending upto the 3rd molar tooth. Submandibular salivary gland rests on the external surface of mylohyoid muscle; only small deeper part extends into the internal surface. Submandibular salivary duct runs about 5 cm between sublingual gland and genioglossus to end in papilla. Lingual and hypoglossal nerves are closely related to gland and duct. Alveolingual sulcus is valley shaped space between tongue and mandibular alveolar bone. Tip of the tongue should be kept upwards to touch the palate to inspect the floor of the mouth (**Fig. 10.20**).

Swelling or ulcer in floor of the mouth should be inspected for its extent, size, shape, margin, edge. Extent from the gum margin, whether crossing midline or not are important especially in carcinomatous ulcer. Unilateral bluish localised swelling may be ranula. *Ranula* extending into the submandibular region across mylohyoid is called as *plunging ranula*. *Sublingual*



Figs 10.18A to D: Carcinoma tongue in different patients. Proper inspection and palpation is essential. Lateral margin is the commonest site – 47%.



Fig. 10.19: Hypoglossal nerve palsy. There is wasting of tongue muscle on same side with tongue deviating towards same side.



Fig. 10.20: Inspection of the floor of the mouth. Tip of the tongue should be kept upwards to touch the palate to inspect the floor of the mouth.

dermoid is in the floor of the mouth *midline* often extends into submental region externally.

Inspection of the Palate

Roof of the mouth is formed by hard palate and soft palate. *Hard palate* is firm anterior part of the roof of the mouth ending opposite 3rd molars anterior to fovea palatine. *Soft palate* is mobile posterior part of the roof of the mouth. Junction between hard and soft palate is called as *vibrating line*. Small rounded elevation of tissue on the midline behind the central incisors is called as *nasopalatine papilla* which is over incisive foramen through which nasopalatine nerve traverses to supply anterior hard palate. Slightly elevated central line is called as palatine raphe. Here mucosa is firmly adherent to underneath periosteum without any fat and so it is harder area of hard palate. Sides of hard palate contain fat and minor salivary glands (there are around 350 minor salivary glands in posterior hard palate). Series of elevations in hard palate are called as *palatine rugae* useful for food positioning and aiding tongue to produce specific sounds. Hard palate is partition between nasal and oral cavity. Anterior 2/3rd is formed by palatine process of maxillae; posterior 1/3rd is by horizontal plates of palatine bones. Anterolateral margins continue with alveolar arches and gums. Posterior margin attaches to soft palate.

Soft palate is redder than hard palate due to its vascularity. There is no bone in soft palate behind vibrating line. Soft palate vibrates or moves. It is mobile muscular fold. It has got anterior and posterior surfaces, superior and inferior margins. Uvula is small fleshy part projecting from centre of the posterior margin of the soft palate. Pair of pits on either side of the centre of the soft palate just behind the vibrating line is called as *fovea palatini* to which palatine mucus glands opens. Side of the uvula has got anterior and posterior folds. Anterior palatoglossal arch contains palatoglossus muscle ends as anterior pillar of fauces (in front of tonsils). Posterior palatopharyngeal arch contains palatopharyngeus muscle ends as posterior pillar of fauces (behind tonsils). Soft palate contains mucus glands and taste buds. Soft palate contains following muscles – tensor veli palati; levator veli palati; musculus uvulae; palatoglossus; palatopharyngeus. All muscles except tensor palati are

supplied through pharyngeal plexus through cranial part of accessory nerve; tensor palati is supplied by the mandibular nerve. General sensory nerves are derived from middle and posterior palatine nerves which are branches of maxillary nerve and from glossopharyngeal nerve. Gustatory special sensations are carried through lesser palatine nerve → greater petrosal nerve → geniculate ganglion of facial nerve → nucleus of solitary tract. Secretomotor fibres are derived from superior salivatory nucleus through greater palatine nerve and lesser palatine nerves. Paralysis of the soft palate (vagus nerve lesions) causes nasal regurgitation of liquids, nasal twang in voice, flattening of palatal arch.

Cleft palate is a congenital defect – of uvula, soft palate or hard palate with nasal septal defect or with cleft lip should be looked for carefully. *Swelling* in the palate may be minor salivary gland tumour. Detailed inspection of such swelling should be done. *Ulcer* palate could be carcinoma/syphilis/tuberculous. Gummatous ulcer is painless with punched out edge and often with perforation. Carcinomatous ulcer is with everted edge; tuberculous is with undermined. It is also important to check *uvular movements* and sensations (**Fig. 10.21**).



Fig. 10.21: Cleft lip and palate in an adult.

Inspection of Tonsils and Fauces

Inspection of tonsils and fauces should be done to look for ulcers/tubercles/growth/leukoplakia, etc. (**Fig. 10.22**).



Fig. 10.22: Inspection of fauces is done using tongue depressor.

Palpation

Palpation of Lip

Both upper and lower lips should be examined. Usually carcinoma lip is nontender initially. Later it becomes stony hard in consistency. Indurated edge is typical. Extent of lesion should be assessed carefully; whether it crosses the midline, whether extends into cheek, angles of mouth are important in deciding the surgical intervention (**Figs 10.23A to 10.24B**). Lesion is held with fingers of one hand and with other hand lip is held to check the mobility. Carcinoma is always fixed. Benign lesions like mucus cyst are mobile. Mucus cyst will be fluctuant and transilluminant. Hunterian chancre is rubbery hard in consistency.

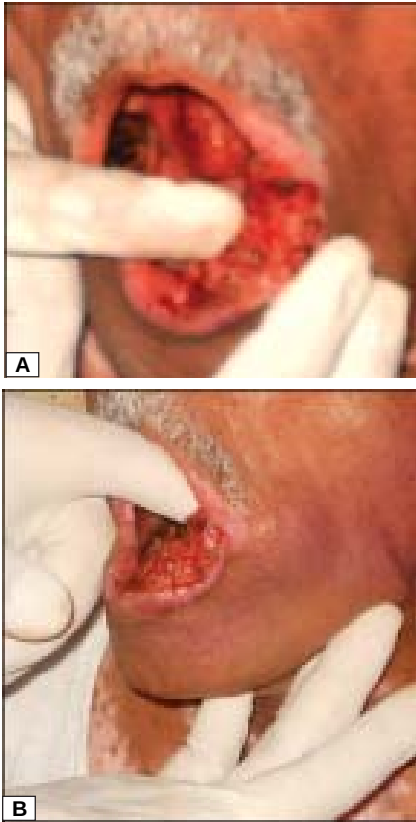
Palpation of Cheek

Cheek should be palpated for any ulcer, swelling. Ulcer due to carcinoma will show induration of edge, base and surrounding area. Its extent should be checked. Posterior extent is important. If it extends beyond retromolar trigone, it means it is advanced. Involvement of soft tissues, mandible, and skin over cheek



Figs 10.23A to C: Examination of lip – methods.

should be checked. *Retromolar trigone* is the anterior surface of the ascending ramus of the mandible. It is triangular in shape with the base being superior and apex lying inferiorly behind the third molar tooth.



Figs 10.24A and B: Palpation of lips carefully to assess the extent of involvement is essential in carcinoma lip.

How much gap is present between growth and alveolar margin should be checked. Other part of the oral cavity should also be palpated. Mandible is palpated using two fingers. Index finger of one hand is placed inside the mouth to feel over the lingual surface of the mandible. Finger of other hand is placed over outer surface of the mandible. Fingers are run along the surface of the mandible to feel tenderness, thickening or any fracture site (features of mandibular involvement by carcinoma) – *bidigital palpation of the mandible*. Mandible is involved by direct extension or through subperiosteal lymphatic plexus which are communicating with oral lymphatics (**Fig. 10.25**).

Palpation of Gums

Bleeding from the gums on palpation is an important finding. It may be due to growth, leukaemia, uraemia, scurvy, epulis. When any lesion is present either



Fig. 10.25: Bidigital palpation of the mandible for tenderness, thickening, irregularity and fracture site should be done in all oral carcinoma – to assess the involvement.

swelling or ulcer its size, shape, extent, tenderness, induration, mobility should be checked.

Palpation of Tongue

Tongue should be palpated with tongue inside because due to contraction of the tongue muscles protruded tongue feels harder mimicking induration. Gummatous ulcer often may be indurated. Tuberculous ulcer is not indurated (It is painful, tender, often multiple). Bleeding on palpation, extent of induration, whether lesion is crossing the midline, tongue movements, floor of the mouth in relation to the lesion should be checked. Entire length of lateral margins should be palpated carefully. Often cheek is retracted using a spatula to palpate the tongue. Recess between lateral base of the tongue and anterior pillar of the fauces is examined (**Fig. 10.26**).

Palpation of posterior third of the tongue is often difficult. Often no growth is visible in this site or only part of the growth is visible. When hyperactive gag reflex is present local anaesthetic spray can be used prior to examination. Patient is asked to open the mouth widely. All left hand fingers of examiner are kept straight and stiff and are pressed firmly over the patient's cheek so that they intervene between upper



Fig. 10.26: Tongue should be palpated with tongue laid within the oral cavity. Otherwise induration is difficult to assess. Protruded tongue will be firm normally while palpation.

and lower teeth. Palpation is done using examiner's right index finger over posterior part of the tongue. Left hand fingers prevent biting of the right examining finger by the patient. By reflex patient may bite only his pushed cheek (**Figs 10.27A to C**).

Palpation of the Floor of the Mouth

It is palpated by asking the patient to put the tip of the tongue on the roof of the mouth with head bending slightly backwards. *Ranula* is an extravasation cyst arising from sublingual or mucus glands. It is smooth, soft, fluctuant and brilliantly transilluminant. When it extends into submandibular region across posterior margin of mylohyoid muscle with cross fluctuation it is called as *plunging ranula*. *Sublingual dermoid* is usually midline swelling in the floor of the mouth with extension outside into submental region. It is smooth, soft, fluctuant but not transilluminant. Carcinoma floor of the mouth is stony hard with indurated edge and base. Mandibular thickening may be felt. It is often fixed.

Palpation of palate: Alveolar abscess is felt as tender fluctuant swelling near alveolar margin. *Gumma* may present as soft swelling in the midline; painless non-tender slightly indurated ulcer in the midline (**Figs 10.28A and B**).



Figs 10.27A to C: Examination of posterior part of the tongue needs special method (See text).

Palpation of Tonsils and Fauces

It should be done in posterior growths of cheek and tongue and in tuberculosis. Surface ulcerations, induration should be looked for. Peritonsillar abscess, carcinoma tonsil, carcinolymphoma of tonsil should be kept in mind (**Fig. 10.29**).



Figs 10.28A and B: Palpation of the floor of the mouth.

Examination of Cervical Lymph Nodes

Cervical lymph nodes should be examined. Submental, submandibular, upper, middle and lower deep cervical and posterior triangle nodes should be examined. Size, shape, mobility, fixity, number should be checked. Both sides should be examined for cervical nodes as lymphatics cross communicate especially in carcinoma tongue and floor of the mouth. All levels should be examined properly (**Figs 10.30A to G**).

Systemic Examination

Eventhough metastatic (blood spread) disease is rare in oral carcinomas, respiratory system examination is important as aspiration pneumonia is common in oral carcinoma especially in carcinoma tongue. Melanoma, lymphoma, rarely aggressive carcinoma can spread to bone, liver through blood. Abdominal and musculoskeletal system examination should be completed (**Fig. 10.31**).

Investigations

Biopsy of ulcer: Edge biopsy is done. Usually two biopsies are taken. If it is on the anterior aspect it can be done under local anaesthesia. Posterior lesions are biopsied under general anaesthesia. Suction apparatus should be used during biopsy. Biopsy area may be apposed using catgut sutures to prevent



Fig. 10.29: Examination of oral cavity should be methodical – lips; gums; cheeks; tongue; floor of the mouth; palate; posterior aspect of the oral cavity.



Figs 10.30A to G: Examination of different levels of neck nodes – Level Ia; Ib; II; III; IV; V; VI.

bleeding. Malignant *squamous cells with epithelial pearls* (Keratin pearls) are the histological features of carcinoma. **Broder's histological grading**—(1) Well differentiated: > 75% epithelial pearls; (2) Moderately differentiated: 50–75% epithelial pearls; (3) Poorly differentiated: 25–50% epithelial pearls; (4) Very poorly differentiated: < 25% epithelial pearls.

Orthopantomogram (OPG): OPG is a must in all oral carcinomas to see mandibular involvement.

Cortical thinning, and bone destruction are looked for. It is plain X-ray mandible showing entire mandible in a single plane. It is a rotational tomogram showing dentition, inner and outer plates of mandible and joints. It is done in jaw tumours, osteomyelitis of mandible, fracture mandible and to see spread from carcinoma oral cavity (**Fig. 10.32**).

Chest X-ray to see bronchopneumonia.
FNAC of cervical lymph nodes.



Fig. 10.31: Respiratory system should be examined for possible development of bronchopneumonia in oral carcinoma especially of tongue.

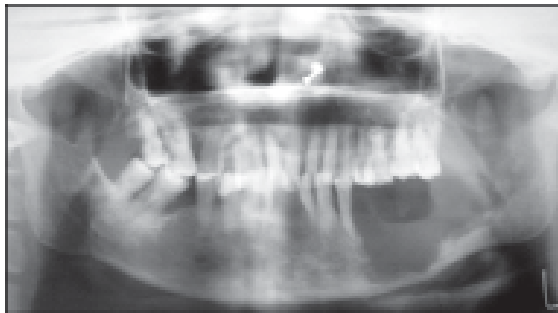


Fig. 10.32: Orthopantomogram is a must to see mandibular invasion in carcinoma oral cavity.

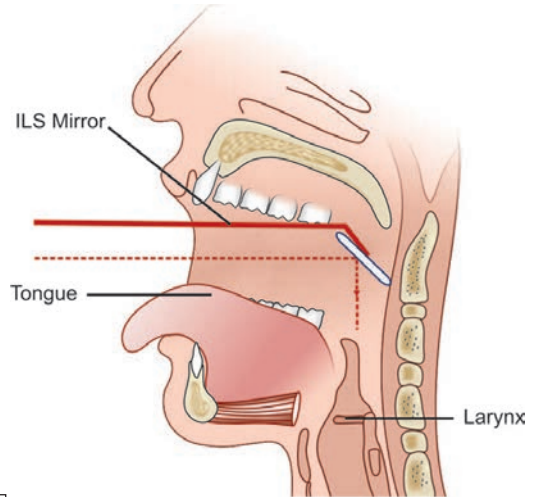
US neck to identify enlarged nodes.

CT scan: It is important in posterior lesions. It also identifies neck nodes, retropharyngeal nodes.

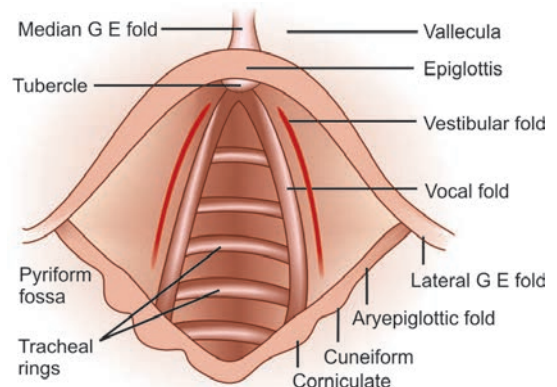
Indirect laryngoscopy/posterior rhinoscopy, direct laryngoscopy, bronchoscopy, are other often needed methods to evaluate the patient (**Figs 10.33A and B and 10.34**).

Ranula (*Rana*—frog, looks like belly of frog)

It is an extravasation cyst arising from sublingual gland and mucus glands of *Blandin and Nuhn* in the floor of the mouth. Occasionally it can occur in submandibular salivary gland also. Initially there is blockage of the sublingual duct causing retention cyst; later



A



B

Figs 10.33A and B: Indirect laryngoscopy to visualise larynx and its parts and view of larynx as seen in laryngoscope.

increased pressure causes rupture of acini leading to extravasation cyst.

Clinical features: It presents as a bluish smooth, soft, fluctuant, brilliantly transilluminant swelling in the lateral aspect of the floor of the mouth (**Figs 10.35A and B**). It often extends into the submandibular region through the deeper part of the posterior margin of mylohyoid muscle and is called as *plunging ranula*. It is cross fluctuant across mylohyoid muscle. Ranula has a delicate fibrous capsule and is lined by a layer of macrophages. It contains clear fluid. It is nontender and laterally placed.

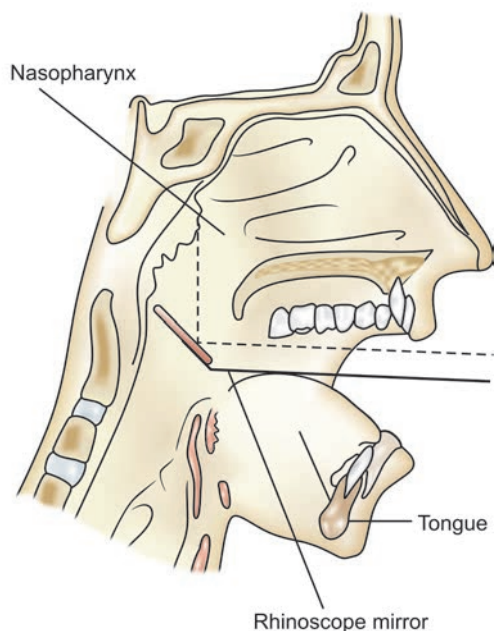


Fig. 10.34: Posterior rhinoscopy.



Figs 10.35A and B: Ranula. It is brilliantly transilluminating swelling.

Sublingual Dermoids

They are *sequestration dermoids* lined by squamous epithelium containing keratin. **Types:** (1) *Median sublingual dermoid*: It is derived from epithelial cell rests at the level of fusion of two *mandibular arches*. It is located between two genial muscles, above the level of mylohyoid muscle. It is a midline swelling which is smooth, soft, cystic, nontender, nontransilluminant (**Fig. 10.36**). (2) *Lateral sublingual dermoid*: It develops in relation to submandibular duct, lingual nerve and stylohyoid ligament. It is derived from *first branchial arch*. It forms a swelling in the lateral aspect of the floor of the mouth.



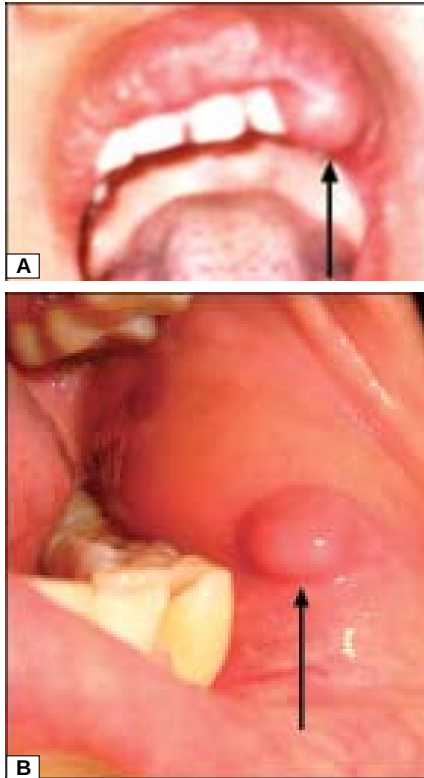
Figs 10.36A and B: Sublingual dermoid. (A) Shows inside swelling (black arrow) extending externally to submental region (white arrow). (B) Magnified view of the inside swelling.

Mucus Retention Cyst

It is due to blockage of the duct of small mucus glands located in the mucous membrane. It is common in inner side of the lower lip or cheek. Cyst develops at any age group. It is painless, slowly progressive, soft, smooth, cystic often transilluminant (but difficult to elicit). It can often rupture spontaneously or get bitten. When its epithelial covering is healthy, it is pale pink in colour with gray glary appearance of visible mucus in the cyst (**Figs 10.37A and B**). If overlying epithelium is damaged, it becomes white scarred and obscure coloured. Overlying mucous membrane is free; it isn't adherent to underneath orbicularis oris or buccinator. Cervical lymph nodes are not enlarged.

Leukoplakia

It is a white patch in the mucosa of the oral cavity that cannot be characterized clinically or pathologically



Figs 10.37A and B: Mucus retention cyst of upper lip and cheek in two different patients.

to any other disease. It is a premalignant condition.

Types: (1) *Homogenous*. (2) *Nodular*—more potentially malignant. (3) *Speckled*—much more potentially malignant.

Clinically the lesion appears as white or grayish coloured, well localised patch in the cheek, tongue, palate or other areas of the oral cavity (**Fig. 10.38**).

Common causes: Smoking, spirit, sepsis, superficial glossitis, syphilis, spices, sharp tooth, susceptibility, pan chewing using areca, tobacco, slaked lime, chronic hypertrophic candidiasis. **Incidence** of leukoplakia in those who smoke or chew pan is 20%, whereas incidence in nonsmokers is 1%. **Incidence** of it turning into malignancy is 2-4%. It increases with age, duration of the pan chewing, smoking.

Histology: *Parakeratosis with widening of rete pegs*.

Histological staging: Acanthosis; Parakeratosis; Widening of rete pegs; Dyskeratosis; Dysplasia; Carcinoma *in situ*.



Fig. 10.38: Leukoplakia cheek.

Erythroplakia

It is red velvety appearance of the mucosa which cannot characterise any recognised condition. It is 17-20 times more potentially malignant than leucoplakia. Histologically *parakeratosis with severe epithelial dysplasia* is the typical feature. It is equal in both sexes. It is common in lower alveolar mucosa, gingivobuccal sulcus and floor of the mouth. It can be homogenous, speckled, and granular or erythroplakia interspersed with leukoplakia.

Oral Submucosal Fibrosis

It is a progressive fibrosis deep to the mucosa of the oral cavity which causes *trismus and ankyloglossia*. The mucosa of cheek, gingivae, palate and tongue shows a *mottled/marbled pallor*. It is common in Asians and Indians.

Etiology: Hypersensitivity to chilli, betel nut, tobacco and vitamin deficiencies (riboflavin) probably alter the collagen metabolism leading to juxtaepithelial fibrosis, epithelial atrophy and dysplasia. 4-7% of oral submucosal fibrosis can turn into *malignancy*. Prevalence in India is 5 per 1000. It is common in middle age; equal in both sexes; incidence is 4-7%. Initial red area turns into superficial ulcers which later forms stiff fibrotic bands and scarring. It is common in buccal mucosa, soft palate and faucial pillars. It is progressive disease showing epithelial hypertrophy, hyperplasia, dysplasia and fibrosis (**Fig. 10.39**).



Fig. 10.39: Oral submucosal fibrosis.

Premalignant Conditions of Oral Cavity

High Risks—Lesions with Definite Risk of Malignant Change

Leukoplakia; erythroplakia; chronic hyperplastic candidiasis—It is common in commissures of the mouth and tongue (**Fig. 10.40**).

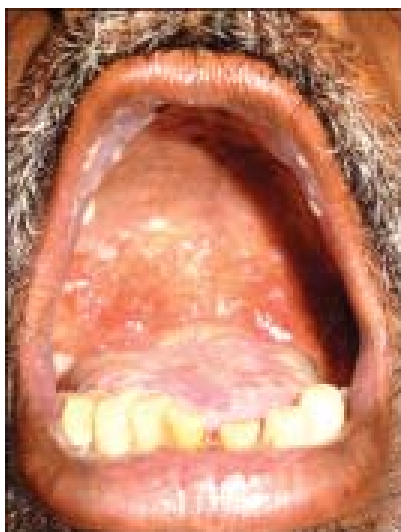


Fig. 10.40: Oral candidiasis—curdy white patches.

Medium Risks – Premalignant but not Associated with Higher Incidence of Carcinoma

Oral submucosal fibrosis; syphilitic glossitis; sideropenic dysphagia (*Sideropenia* is iron deficiency

without anaemia); or Plummer-Vinson syndrome. Sideropenia is common in Scandinavian females. It causes atrophy of epithelium and becomes potentially malignant. Proper iron therapy controls the disease and reduces the risk.

Equivocal Risk Lesions

Oral lichen planus, dyskeratosis congenital, discoid lupus erythematosus.

General Features of Oral Carcinoma

Oropharyngeal cancer is the most common cancer – 40% in Indian subcontinent. In western countries it accounts for 4% only. *Risk factors*—tobacco and related products; alcohol; areca nut; human papilloma virus; Epstein Barr virus; Paterson-Kelly syndrome; nutritional deficiency. Patient may develop a second primary (15%) in the oropharynx in different site at the same time or within 6 months of the existing primary (*synchronous*—4% prevalence; 20% of second primaries) or after 6 months of first primary (*metachronous*—80% of second primaries). Metachronous second primary is more common than synchronous second primary and it usually occurs in 2 years. In India cheek is the commonest site, then tongue, floor of the mouth, palate and lips. In western countries tongue, floor of the mouth, lip and cheek is the order of occurrence. *Problems with oral carcinoma*: Upper airway obstruction especially posterior growths; bronchopneumonia; feeding difficulties; malnutrition; infection; torrential bleeding due to erosion of vessels like lingual; fixity of secondaries; fungation; disability and psychological discomfort (**Fig. 10.41**).

Carcinoma Cheek

Squamous cell carcinoma is the most common carcinoma of the cheek. Occasionally it can be adenocarcinoma arising from the minor salivary glands or mucus glands. It may be also rarely melanoma.

Precipitating Factors

All ‘S’—Smoking, Spirit, Syphilis, Sepsis, Sharp tooth, Spices. It is common in Chutta smokers (Tobacco enrappd in a tobacco leaf). Chutta carcinoma is



Fig. 10.41: Carcinoma of lip, cheek and tongue. Patient was earlier (3 years early) operated for carcinoma cheek. Now metachronous growths appeared in lip, cheek and tongue.

common in Andhrapradesh and Orissa. Pipe smoking (buccal carcinoma); snuff use (floor of the mouth); different mouth washes may be other aetiologies.

Premalignant Conditions

Leucoplakia, erythroplakia, submucosal fibrosis, hyperplastic candidiasis. *Betel nut chewing* (*Pan*, with pan quid kept in cheek pouch for a long time) is an important causative factor of carcinoma cheek. *Types:* Ulcerative; proliferative (exophytic); verrucous.

Verrucous Carcinoma

It occurs as a superficial proliferative exophytic lesion with minimal deep invasion. Lesion has got white, dry, velvety or warty, keratinised surface. It is of low grade, very well differentiated squamous cell carcinoma, which is locally malignant without any lymphatic spread. It is a curable malignancy. It is common in females. It may be related to human papillomavirus. It is often multicentric. Invasive carcinoma also may develop in other sites.

Biological Behavior of Carcinoma Cheek

Carcinoma cheek is common in posterior half of cheek than anterior. It spreads into the deeper plane to involve

buccinator, pterygoids; into the retromolar trigone, base of the skull, pharynx. It spreads outwards to involve the skin causing fungation, ulceration, orocutaneous fistula formation (**Fig. 10.42**). Mandible is commonly involved either by direct extension or through subperiosteal lymphatic plexus which communicates freely with oral lymphatics. Lymph nodes commonly involved are submental, submandibular, deep cervical and often lateral pharyngeal groups. Nodal spread is observed in 50% of cases. Infection of the tumor area and soft tissues around is common, causing fever, foul smelling ulcer, halitosis. Respiratory infection is common in these patients. Once tumor extends into the retromolar region, soft palate, pharynx, dysphagia will occur.



Fig. 10.42: Carcinoma of cheek with proliferative lesion extending to skin with sinus/possibly orocutaneous fistula.

Clinical Features

Ulcer in the cheek which gradually increases in size in a patient with history of chewing pan, and smoking. *Pain* occurs when it involves the skin, bone or if secondarily infected. Referred pain into the ear signifies involvement of lingual nerve. *Involvement of retromolar trigone* indicates that it is an advanced disease, as the lymphatics here communicate freely with the pharyngeal lymphatics. *Everted edge, induration* are the typical features of the ulcer. Mandible is examined bidigitally, for thickening, tenderness, and sites of fracture. *Trismus and dysphagia* signifies involvement of pterygoids, or posterior extension. Occasionally it may extend into the *upper alveolus* and to the *maxilla* causing swelling, pain and tenderness. Submandibular lymph nodes and upper deep cervical lymph nodes are involved which are



Fig. 10.43: Carcinoma of cheek involving skin and soft tissues – locally advanced malignancy.

hard, nodular, and initially mobile but later get fixed to each other and then to deeper structures. Once lymph nodes get fixed it may infiltrate into hypoglossal nerve (tongue will deviate towards the same side), spinal accessory nerve (defective shrugging of shoulder) and cervical sympathetic chain (*Horner's syndrome*). Compression over external carotid artery causes absence of superficial temporal artery pulsation. Eventually it causes fungation and bleeding from major vessels—*carotid blow out* (**Figs 10.43 and 10.44**).



Fig. 10.44: Recurrent carcinoma cheek. Earlier patient has undergone surgical wide excision. Note the old scar.

Staging (for all Oral Carcinomas)

TNM Staging.

- T₁ Tumour size < 2cm
- T₂ Tumour size 2-4 cm
- T₃ Tumour >4cm
- T₄ Tumour is of any size involving bone, soft tissues, muscles.
- N₁ Lymph node size <3 cm

- N₂ Lymph node size 3-6 cm or bilateral lymph nodes; N_{2a}—single node 3-6 cm size on same side; N_{2b}—multiple nodes 3-6 cm size on same side; N_{2c}—bilateral or opposite nodes enlarged upto 6 cm size.

- N₃ Lymph node size > 6cm.

Features of Advanced Carcinoma Cheek

Involvement of retromolar trigone; extension into the base of skull and pharynx; fixed neck lymph nodes; extension to the opposite side.

Carcinoma Lip

It is common in men; common in old age; common in *lower lip* (90%) (**Fig. 10.45**); upper lip 5-10%; less common in Negroes; common in white Caucasians. It is commonly due to exposure to sunlight (ultraviolet rays); common in pipe smokers; initially starts as a red, granular dry lesion which eventually gets ulcerated and forms an ulceroproliferative lesion. Occasionally



Fig. 10.45: Carcinoma lower lip

it occurs at the angle of mouth. Often carcinoma of lip is an extension from carcinoma of cheek. It is slowly progressive tumour; it spreads to submental nodes and later to other neck nodes on both sides; usually it is a *well differentiated squamous cell carcinoma*.

Causes: UV rays, smoking, cheilitis, solar keratosis, papilloma, leukoplakia, tobacco chewing, Khaini chewers (tobacco + lime); agriculturist who are exposed to sunlight—*countryman's lip*.

Clinical features: Nonhealing progressive painless ulcer/ulceroproliferative lesion; offensive discharge; proliferative with everted edge; stony hard in consistency; cannot be moved separately from lip; indurated edge and surrounding area; bleeds on touch; palpable *hard* submental/submandibular/upper deep cervical nodes.

Differential diagnosis: Keratoacanthoma, basal cell carcinoma, minor salivary gland tumours.

Carcinoma Tongue

Incidence is equal in both sexes. Presently its incidence is increasing in females due to increase in number of female smokers.

Etiology: Leucoplakia, erythroplakia, all 'S's' mentioned earlier, premalignant conditions mentioned earlier.

Types: Gross: (1) Papillary; (2) Ulcerative or ulceroproliferative; (3) Fissure with induration; (4) Lobulated, indurated mass.

Histologically: Squamous cell carcinoma—commonest; adenocarcinoma may be from minor salivary glands or mucus glands; melanomas. **Sites:** *Lateral margin*—commonest—47-50% (**Fig. 10.46**); posterior third—20%; dorsum—6.5%; ventral surface—9%; tip—10%. It is common in India and France.

Clinical Features

Painless ulcer/swelling in the lateral margin (commonest site) of the tongue which becomes painful later (*Florid, painless, friable, bleeding, everted edge, sloughing yellow gray floor, serosanguinous discharge, induration of edge and base and is extending into surrounding area* are the features). *Pain* in the



Fig. 10.46: Carcinoma tongue is more common in lateral aspect of the tongue.

tongue due to infection or ulceration or due to the involvement of lingual nerve (pain is referred to ear); pain on swallowing, in case of carcinoma of posterior third of tongue; *excessive salivation* (It is due to irritation of nerves of taste buds and ankyloglossia causing difficulty in swallowing saliva); *dysphagia* either due to fixed tongue or due to the involvement of genioglossus or growth in the posterior third of the tongue; *visible ulcer* in anterior two thirds of tongue with raised everted edge, area of induration is more extensive than visible ulcer area; ulcer may cross the midline or extend into alveolus, floor of the mouth, mandible; bleeds on touch; growth or ulcer in posterior third, is usually *not visible*. *Ankyloglossia*; inability to articulate; *foetor oris (halitosis)* due to infection and necrosis in the oral cavity; *change in voice* occurs in posterior third tumours. Tumour in posterior third area is more aggressive. Indirect laryngoscopy is often needed to visualise posterior third of the tongue. *Lymph nodes* may be palpable in the neck which are hard, nodular and may get fixed in advanced stages. Features of bronchopneumonia may be present. **Spread of carcinoma tongue: Local spread:** In case of anterior two thirds of tongue, the spread occurs to genioglossus muscle, floor of the mouth, opposite side and mandible. In case of posterior third of tongue it spreads locally to tonsil, side of pharynx, soft palate, epiglottis, larynx and cervical spine. **Lymphatic spread:** From tip of tongue it spreads to submental nodes. From lateral margin it spreads to submandibular lymph nodes and later to upper and middle deep cervical lymph nodes. Lymphatics in the tongue are freely communicating,

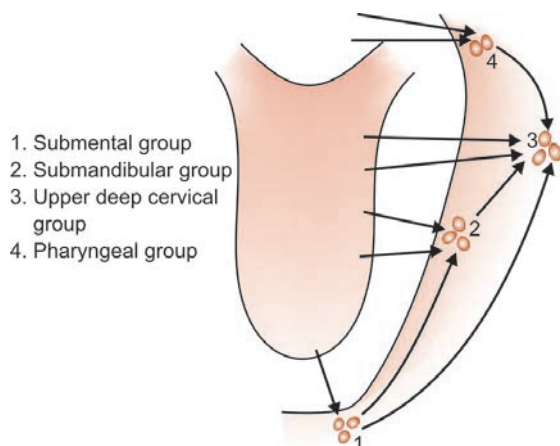


Fig. 10.47: Lymphatic drainage of tongue.

and so involvement of bilateral neck lymph nodes is common. From posterior third it spreads to pharyngeal nodes and upper deep cervical lymph nodes. Posterior third growths can show blood spread (**Fig. 10.47**).

Terminal Events in Advanced Cases

Aspiration pneumonia; erosion of major vessels by primary tumour (lingual artery) or by secondaries in neck (carotid artery); cachexia; laryngeal oedema; asphyxia; starvation due to inability to swallow.

Carcinoma of Posterior One-Third / Base of the Tongue

Here lesion may remain asymptomatic for long time. Clinically it may be missed easily. Earlier symptoms are features mimicking sore throat and throat discomfort. Dysphagia and change in voice (*hot potato voice*) occurs later. Referred pain in the ear, bleeding from mouth, visible mass in posterior third of tongue is late local features. Induration on palpation in posterior third tongue is diagnostic of the carcinoma. As posterior third tongue has got abundant lymphatics which cross communicates on either side, lymph node spread is common (70%). Bilateral nodal spread is common. Massive nodes and involvement of jugulo-digastric node are also common. Infiltration into the tongue muscles like genioglossus, epiglottis, pre-epiglottic space, tonsillar pillars and hypopharynx are

common. Carcinoma posterior third of the tongue is often poorly differentiated and so carries poor prognosis. Blood spread can occur into bones, liver and lungs in posterior third cancers. Palpation under anaesthesia gives better idea about the tumour, its spread and also allows the biopsy. CT scan is always needed to plan the staging and therapy. Lymphoepithelioma and transitional cell carcinoma can occur in posterior third tongue (rarely).

Carcinoma Floor of the Mouth

It is usually aggressive tumour. It is rare in India. It is 2nd common site of oral carcinoma (SCC) in western countries. It invades hyoglossus, mylohyoid, genioglossus and anterior mandible early. Trismus, ankyloglossia, mandibular spread is common. Bilateral neck nodes are commonly involved (**Fig. 10.48**). Prognosis is poor and also has poor cosmetic results.

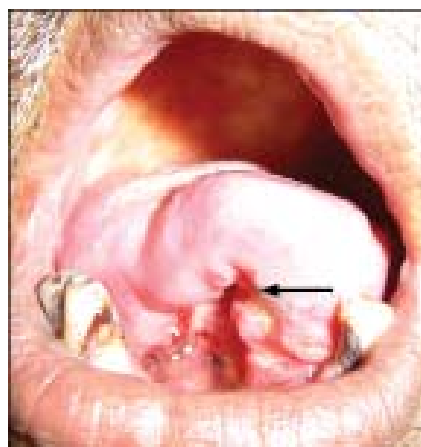


Fig. 10.48: Carcinoma floor of the mouth.

Carcinoma Alveolus

It is squamous cell carcinoma arising from gums. It is common in males. It is common in India. It is commonly due to tobacco/pan chewing. Features and precipitating factors are similar to other oral carcinomas. There will be invariable bone involvement by direct extension. Nodal spread is also common.

Carcinoma of Hard Palate

Squamous cell carcinoma in the hard palate is common in males; common in *reverse smokers* (Rolled tobacco

leaf – Churat); due to repeated thermal injury. It spreads to periosteum, bone, maxilla, sinus or nose. Ulcerated raised everted edge with induration and fixity is the presentation. Upper deep cervical nodal spread occurs in 25% of patients. Hard palate is the common site for minor salivary gland tumours (**Fig. 10.49**). They are commonly malignant; common is adenoid cystic type; single solid smooth swelling with ulcer over the summit is the presentation. Edge biopsy, FNAC of nodes; CT scan of neck and base of skull are the needed investigations.



Fig. 10.49: Carcinoma of hard palate.

Laryngeal Carcinoma

Aetiology: Smoking, tobacco, alcohol intake, occupational/industrial exposure to chemicals like mustard gas, asbestos, benzopyrones, petroleum products, previous radiation, genetic—Russians develop familial laryngeal cancers, papillomavirus, Herpes simplex virus, EB virus, keratosis, malnutrition.

Incidence: Squamous cell carcinoma is commonest (95%); common in males (10:1); common in 5th/ 6th decade.

Types: Ulcerative; Proliferative.

Anatomical types: Supraglottic (25%): It arises from infrahyoid part of epiglottis, ventricles, and arytenoids. It spreads to neck lymph nodes early (40%) due to rich lymphatics in this area. Throat pain, dysphagia,

palpable neck nodes and referred pain are common features. Hoarseness of voice, loss of weight, respiratory obstruction, and halitosis are late features. Carcinoma in epiglottis causes bilateral nodal spread. Local spread occurs to vallecula, base of tongue and pyriform fossa. **Glottic (65%):** It is the commonest type. It begins from upper part or free edge of vocal cords (mid or anterior) often extending 10 mm below. Lymphatic spread is slow (only 4%) as this area has got least lymphatics. Opposite vocal cord can involve as *kiss cancer*. Vocal cord mobility is unaffected in early cases. Vocal cord fixation signifies spread to thyroarytenoid which is a poor prognostic sign. It presents very early due to hoarseness of voice. Eventual cord fixation causes stridor. Locally it spreads anteriorly to anterior commissure, posteriorly to vocal process and arytenoids, above to ventricle and false vocal cords, below to subglottis. **Subglottic (2%)** cancer is less common involving undersurface of true vocal cords and subglottic space. It spreads to deep cervical and paratracheal nodes (20%). Upward spread is rather late and so hoarseness is not an early symptom in this type. It can spread through cricothyroid membrane or thyroid gland (**Figs 10.50 and 10.51**).

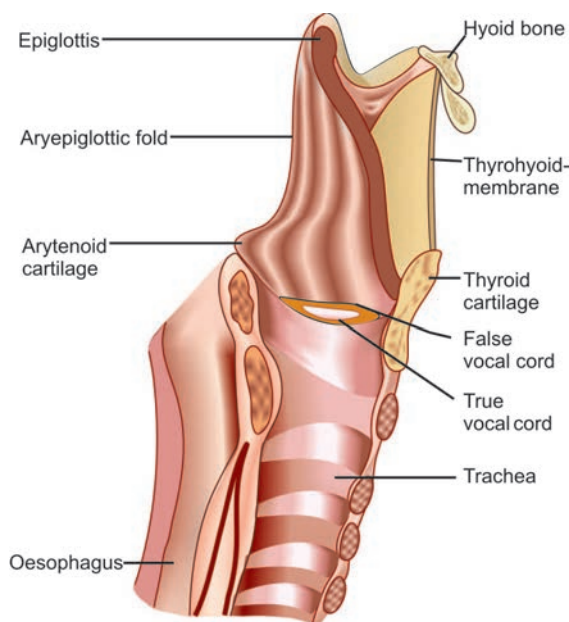


Fig. 10.50: Anatomy of larynx showing supraglottic, glottic and subglottic regions.

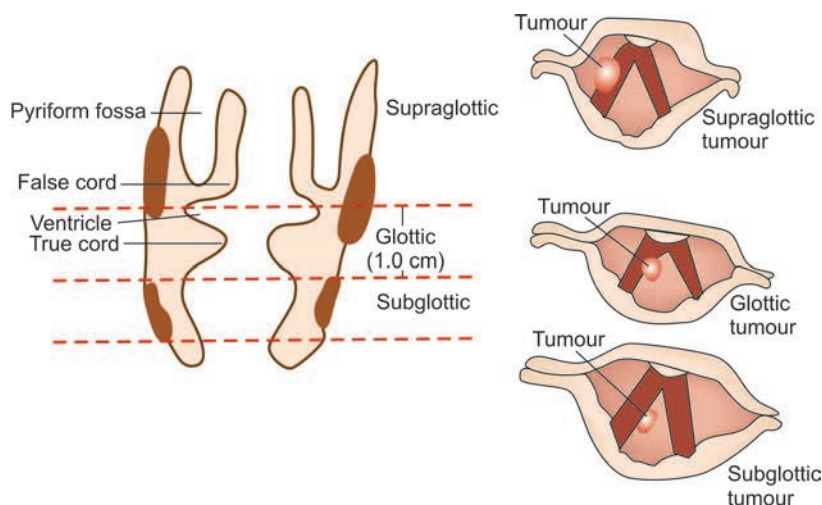


Fig. 10.51: Laryngeal carcinoma types. Note the typical sites. Glottic is the commonest site. Next is supraglottic. Subglottic is rare.

Note: In Indian subcontinent supraglottic tumours are more common than glottic. Glottic type is common in western countries. Fixation of cords is due to involvement of thyroarytenoid muscle or cricoarytenoid joint.

Clinical features: Hoarseness of voice; pain and discomfort; cough, dyspnoea, stridor, dysphagia in late cases; bloody sputum; palpable neck nodes, which eventually get fixed; absence of laryngeal crepitus. It is common in males—10:1.

Investigations: ILS (Indirect laryngoscopy); direct laryngoscopy and biopsy; CT neck—*very useful investigation*; chest X-ray; FNAC of lymph node; microlaryngoscopy in small lesions to identify and to have proper biopsy; Toluidine blue staining to stain early superficial cancers which facilitate the accurate biopsy; Hopkin's endoscopy; Flexible, fiberoptic laryngoscopy.

Nasopharyngeal Carcinoma

Nasopharynx lies above the level of the soft palate which separates it from oropharynx below. It is also called as post-nasal space or epipharynx. Eustachian tube opens on its anterolateral wall. Fossa of Rosenmuller is located above and behind the opening of the Eustachian tube as a small depression.

Clinical features: Epistaxis, nasal speech, post-nasal discharge and nasal obstruction; pain in the ear with unilateral deafness due to compression of Eustachian tube with fluid collection in the middle ear; elevation and immobility of soft palate on the same side; pain in the area of distribution of trigeminal nerve due to direct infiltration of the nerve at foramen lacerum; palpable secondaries in upper deep cervical lymph nodes (70%).

Trotter's triad: Unilateral deafness; immobile elevated soft palate; pain in the distribution of trigeminal nerve. Nasopharyngeal carcinoma is common in China, Taiwan, Hong Kong and Mongolia. It is rare in USA. In India it is common in north east region. It is common in males (2:1). It may be related to Epstein-Barr virus. It is commonly squamous cell carcinoma (85%). Lymphoma, minor salivary tumors and sarcoma are other malignancies that can occur rarely in nasopharynx. Lymphoepithelioma of nasopharynx is called as *Schminke/Regaud tumour*. Carcinoma can be of proliferative, ulcerative, and infiltrative types. Commonest site is fossa of Rosenmuller in lateral wall of pharynx. It is three times common in males. *HO's triangle* in supraclavicular fossa (bounded by medial and lateral ends of clavicle and point where neck meets the shoulder) is the site where metastatic nodes commonly exist in nasopharyngeal carcinoma. In 50% of cases nodal involvement is bilateral. Often cervical

lymphadenopathy may be the first presentation. Clinical features may be nasal, otogenic, ophthalmoneurogenic (involving most of the cranial nerves with facial pain, squint, diplopia, exophthalmos, and ophthalmoplegia), jugular foramen syndrome (cranial nerves IX, X, XI spread), nodal spread and distant spread to bones, lungs and liver. Unilateral serous otitis media may be the only presentation.

Benign Tumours of the Tongue

Papilloma; fibroepithelial polyp; haemangioma and lymphangioma; neurofibroma; lipoma; granular cell myoblastoma are benign tumours of the tongue (**Fig. 10.52**).



Fig. 10.52: Papilloma of tongue – benign tumour. It can be premalignant. It is firm, well localised swelling which may bleed on touch. It needs excision.

Differential Diagnosis for Tongue Ulcers

Dental ulcers: Jagged tooth or denture causing mechanical irritation of the margin or undersurface of the tongue causing elongated, erythematous, painful ulcer having slough at the floor surrounded by hyperaemia.

Aphthous ulcers: They are usually single but can be multiple, small painful ulcers in the tip/undersurface/ anterior part of the dorsum of the tongue having whitish floor, yellow margin and hyperaemic zone around. It is common in females; often familial. It can be recurrent.

Ulcers in Lichen Planus

Syphilitic ulcers: Extragenital chancre occurs in the tongue as an ulcer which is painless, rubbery hard with thick crust covering; ulcer leaves a fine superficial scar while healing. Shotty discrete lymph nodes in the neck are common – *primary syphilis*. In *secondary syphilis* multiple shallow ulcers are found on the ventral surface and lateral margins of the tongue; mucus patches on the dorsum and tonsillar pillars; Hutchinson's condyloma (warts) are seen on the middle of the dorsum of the tongue. In tertiary syphilis gummatous ulcer is present on the midline of dorsum of tongue which is punched out, deep and nontender.

Tuberculous ulcers: They are multiple undermined ulcers seen at the margin, tip or dorsum of the tongue. They are usually painful when located over the dorsum of the tongue. Pulmonary tuberculosis or laryngeal tuberculosis can occur.

Malignant ulcers are nothing but carcinomatous ulcers.

Post pertussis ulcer occurs after whooping cough infection over the frenulum on the ventral surface of the tongue.

Chronic nonspecific ulcer occurs without any specific aetiology which is not painful but often indurated; occurs over anterior 2/3rd of the tongue; mimics carcinoma of tongue.

Stomatitis

It is a general term used for inflammation of the entire lining of the mouth often including tongue.

Causes of stomatitis: *Local causes:* Sharp teeth, poor fitting dentures, smoking, infections like Herpes virus, candida, and Vincent's angina, trauma either due to mechanical, chemical, thermal or X-rays. *General causes:* Haematological - Anaemia, agranulocytosis, purpura, leukaemia; Vitamin deficiencies—Scurvy (Vitamin C), sprue, coeliac disease, pellagra, pernicious anaemia, kwashiorkor; tuberculosis; advanced carcinoma; drugs like phenobarbitone, phenytoin; lead/mercury/bismuth poisoning; syphilis infection.

Infective stomatitis can occur either by opportunistic (*facultative*) organisms like normal commensals (normally existing organisms causes infection when patient's defense mechanism is reduced)—streptococci,

staphylococci, Vincent's organisms or by *true pathogens*.

Catarrhal stomatitis: It is associated with acute upper respiratory tract infection and acute fevers. The entire mucous membrane of the oral cavity; becomes oedematous and red; small ulcers may coalesce to form typical ulcerative stomatitis.

Aphthous stomatitis: It is formation of small painful tender vesicles of unknown aetiology with hyperaemic base which eventually breaks forming small white circular deep painful ulcer. It is common in cheek, lips, floor of the mouth, soft palate. It is common in females. It heals in 2 weeks. Recurrent solitary aphthous ulcers are also common.

Monilial stomatitis (Oral thrush): Infection of gastrointestinal tract by *Candida albicans* is common in children, debilitated patients, immunosuppressed individuals like HIV patients, patients on cancer chemotherapy or antibiotic therapy. Initially small red patches appear on the mucosa of cheek and tongue which turns curdy white due to desquamated oedematous epithelium with contaminated fungus. These lesions are painful with excessive salivation. Associated thrush in the pharynx, oesophagus is common causing dysphagia also.

Ulcerative stomatitis (Vincent's angina): It is caused by anaerobic gram negative *Borrelia vincentii* (spirochaete) and *Fusiformis fusiformis* bacteria (rod shaped) initially causing severe gingivitis later Vincent's stomatitis. Swollen inflamed, painful, peppered gums with small ulcers covering yellow slough is typical. Later similar lesions appear in cheek, tonsils, fauces. Features are gum bleeding, foetor oris, ill look, toxicity, fever, loss of appetite, enlarged tender neck nodes.

Cancrum oris (Noma) (Fig. 10.53): It is an infective gangrene as a result of severe form of Vincent's acute ulcerative stomatitis and gingivitis seen in children who are malnourished and often in patients with measles and leukaemia; begins in lips, cheek, soft tissues, bone, skin with extensive tissue destruction, ischaemic necrosis; toxæmia, anorexia, pyrexia; excessive salivation, fetid odour; a rare condition nowadays but carries high mortality.



Fig. 10.53: Cancrum oris involving gingiva and lower lip extensively with plenty of maggots in it.

Angular stomatitis (Cheilosis, Perleche): They are inflamed red brown fissures at the corners of the mouth probably due to dribbling of saliva at the corners; common in edentulous; Perleche is common in children who rub or lick the corners of their mouth (*Perleche* means lick in French). Condition does not extend to adjacent mucous membrane and heals without scarring (**Fig. 10.54**).



Fig. 10.54: Severe infection of lip (Cheilitis), angle of mouth. Patient is also having severe stomatitis.

Syphilitic Lesions of Oral Cavity

Syphilis is rare nowadays. Primary syphilis shows *Hunterian chancre* in the lip and tongue. It is similar to that occur in genitalia. Flat pink painless initially macule becomes hemispherical papule with breakage of superficial mucosa causing ulcer with thick crust and rubbery base; a fine superficial scar is formed when ulcer heals; neck lymph nodes may be enlarged. *Rhagades* are radiating cracks developing in secondary syphilis which extends to mucous membrane which on healing leaves a fine scar. Gray white pearl coloured *mucous patches* develop on the inner side of lips, cheeks and pillars of tonsils, often reaching upto 2 cm size; with desquamated oedematous epithelium as white patch. It is contagious and often presents as sore throat. Linear ulcers are seen in faucial pillars covered with transparent glistening mucus or white boggy epithelium looking like 'snail track' ulcers.



Fig. 10.55: Tongue tie: Typical look (for detail refer Page No. 231) (Courtesy Dr Sathish, MCh, Plastic Surgeon, Mangalore).

11

Examination of Jaw

There are two jaws—upper and lower. Upper jaw is formed by maxilla and lower jaw by mandible.

Mandible is the largest, and strongest bone of the face. It has got horse shaped body with two rami projecting upwards from its posterior ends. Outer surface of body contains symphysis menti, mental protuberance, mental foramen, oblique line, incisive fossa. Inner surface contains mylohyoid line, submandibular fossa (below), sublingual fossa (above), genial tubercles. Upper alveolar border bears sockets for teeth. Quadrilateral ramus has got medial and lateral surfaces, anterior, posterior, upper and lower borders and coronoid and condyloid processes. Medial surface of ramus contains mandibular foramen above the centre of the ramus near the occlusal surface of teeth (transmits inferior alveolar nerve and vessels across mandibular canal to mental foramen on the outer surface); lingula (bony projection of mandibular foramen gives attachment to sphenomandibular ligament); mylohyoid groove (medial pterygoid is inserted below and medial to this groove). Lateral surface of ramus is flat (attachment of the masseter muscle). Upper border of ramus forms curved mandibular notch; lower border containing angle of mandible is the continuation of the base of mandible; posterior border of ramus is thick; anterior is thin. Anterior projection is called as *coronoid process* (temporalis is inserted on its apex and medial surface); posterior projection is called as *condyloid process*. Condyloid process is strong with expanded upward head which articulates with temporal bone to form temporomandibular joint. Anterior part of neck of condyloid process has got pterygoid fovea for the insertion of lateral pterygoid muscle. Oblique line on the outer surface of the body gives origin to buccinator; mylohyoid line on the inner surface of body gives origin to mylohyoid and superior constrictor muscles; upper

genial tubercle gives origin to genioglossus and lower to geniohyoid; digastric fossa gives origin to anterior belly of digastric muscle. Investing layer of deep fascia and platysma is attached to lower border (base) of the mandible. Masseteric vessels and nerve passes through mandibular notch; mental foramen transmits mental vessels and nerve; inferior vessels and nerve passes through the mandibular canal; mylohyoid vessels and nerve are related to mylohyoid groove; lingual nerve is related on the medial surface; auriculotemporal nerve is related to medial side of the neck of the mandible. Mandible is the second bone to ossify in the body after clavicle. Ossification centre (*only one centre*) appears in 6th week of intrauterine life, one on each side near mental foramen. Entire *body* ossifies from *membrane* except only one near incisor teeth; ramus *above the mandibular foramen* ossifies from *cartilage*. Site at canine socket is weak and is the *commonest site of fracture* which may involve inferior alveolar nerve causing neuralgic pain and loss of sensation over the distribution of mental nerve (**Fig. 11.1**).

Maxilla is the 2nd largest bone of the face. Two maxillae form upper jaw. Each maxilla has got body, 4 processes – frontal, zygomatic, alveolar and palatine. Body is pyramidal in shape with base medially at nasal surface; apex laterally at zygomatic process. Body has got 4 surfaces – anterior/facial; posterior/infratemporal; superior/orbital and medial/nasal. Anterior facial surface gives attachments to many muscles of facial expression. Infraorbital foramen transmitting the infraorbital vessels and nerve is above the canine fossa. Posterior surface forms the anterior wall of infratemporal fossa. Maxillary tuberosity gives origin to superficial head of medial pterygoid muscle. Anterior wall of the pterygopalatine fossa is above the tuberosity – grooved by maxillary nerve. Superior surface is

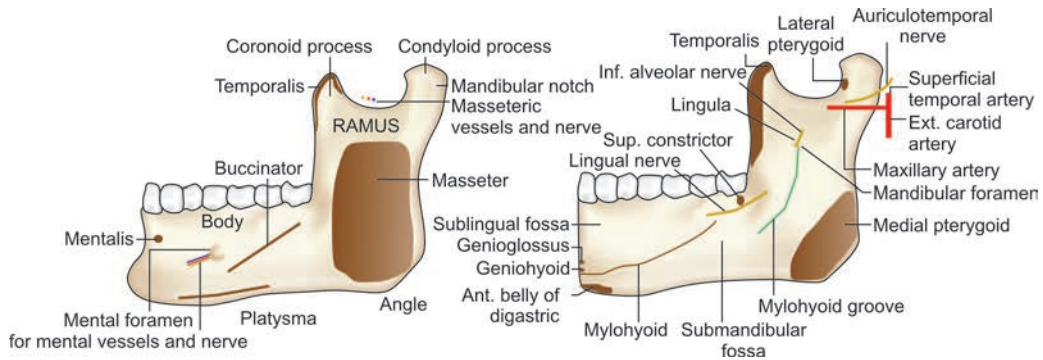


Fig. 11.1: Anatomy of mandible showing attachments and relations.

orbital surface forming floor of the orbit. It is related to lacrimal crest, inferior orbital fissure, nasolacrimal canal (contains nasolacrimal duct), infraorbital groove, inferior oblique muscle. Medial nasal surface forms the lateral wall of the nose. Posterosuperiorly maxillary sinus opening, and maxillary hiatus is present. Below inferior meatus of nose is present. Behind hiatus, there is greater palatine canal containing greater palatine vessels, anterior, middle and posterior palatine nerves. In the alveolar process canine socket is deepest; molar sockets are widest with each having 3 minor sockets. Palatine process is thick horizontal medial projection forming roof of the mouth and floor of the nasal cavity. Two palatine processes, one on each side forms the anterior 3/4th of the bony palate which articulates with the horizontal plate of palatine bone. Greater palatine vessels and anterior palatine nerves are present posteromedially. Maxilla articulates laterally with 1 bone – zygomatic; superiorly with 3 bones – nasal, frontal, ethmoidal; medially with 5 bones – ethmoid, inferior nasal concha, vomer, palatine and opposite maxilla. *Maxillary sinus* is the pyramidal shaped cavity inside the body of maxilla with base medially and apex towards zygomatic process. Its roof is floor of the orbit. Its floor is alveolar process of maxilla. It is $3.7 \times 3.7 \times 2.5$ cm in size. Maxillary sinus is *first sinus* to develop. Maxilla ossifies from **membrane** from 3 centres. One for maxilla proper is above the canine fossa during 6th week of intrauterine life. Two for premaxilla – one just above the incisive fossa at 6th week; another paraseptal is at 10th week.

Temporomandibular joint is a condylar synovial joint. Upper temporal articular surface articulates with lower head of the mandible. Joint is covered with

fibrocartilage with an intra-articular disc inside dividing the joint into upper and lower parts. Fibrous capsule, lateral temporomandibular ligament, stylo-mandibular ligament and sphenomandibular ligaments are the supports. Sphenomandibular ligament is related to lateral pterygoid, auriculotemporal nerve, maxillary artery, chorda tympani and pharynx. Laterally joint is related to parotid and temporal branches of facial nerve; medially tympanic plate, internal carotid artery, sphenomandibular ligament and related structures; below maxillary vessels; behind parotid, external auditory meatus, superficial temporal vessels, auriculo-temporal nerve.

Movements: *Depression* (mouth opening) is by lateral pterygoid mainly (gravity muscle) supported by digastric, geniohyoid and mylohyoid; *elevation* is by masseter, temporalis and medial pterygoid (antigravity muscles); *protrusion/protraction* by both lateral and medial pterygoids; *retraction* by posterior fibres of temporalis. *Lateral/side-to-side* movement is by same side lateral pterygoid and opposite side medial pterygoid.

History taking begins with:

Name:

Age:

Occupation:

Address:

History

History of present illness: History of trauma and method of injury should be asked for. Blood stained saliva after trauma suggests compound fracture

especially in mandible as mucoperiosteum is adherent. Pain, swelling in the floor of the mouth (haematoma), difficulty in speech and swallowing, difficulty in moving the jaws are the other history to be asked. History of swelling, its duration, progression of swelling; History of pain, its nature, severity, progression; History of nasal block, nasal discharge, epistaxis; History of visual disturbances (diplopia, eyeball protrusion); History of swelling in the oral cavity; History of headache over the sinuses are other matters to be asked for. Referred pain in the ear can occur though auriculotemporal nerve. Maxillary sinus tumours can present with swelling, nasal problems, visual disturbances, headache. History of ulcer, swelling in the alveolus, palate or gums should be asked. History of epiphora suggests blockage of nasolacrimal duct causing constant overflow of tears. History of bleeding gums, purulent nasal discharge suggests maxillary antral sepsis (empyema); history of caries teeth, persistent severe neuralgic pain are also important. Tumour may invade especially maxillary division of trigeminal nerve causing severe pain. History of swelling in the neck suggests cervical lymph node enlargement suggesting neoplastic or inflammatory pathology. Its duration, progress, presence of pain should be asked.

Past history: Earlier history of similar complaints; treatment of sinus pathology, surgeries done earlier for similar condition; response to treatment should be asked.

Personal history: Alcohol intake, smoking, tobacco chewing history are important points to be noted.

General Examination

Anaemia, clubbing, pulse, cyanosis, lymphadenopathy, blood pressure should be checked.

Local Examination of Jaw

Inspection

Inspection of outer surface of the maxilla, and mandible is done for swelling, ulcer, skin oedema; discharging sinus (due to dental infection or osteomyelitis of the bone or due to malignancy or due to previous radiotherapy or due to recurrent tumour), upper and lower

lips should be everted to examine jaw properly. Nasal cavity should be inspected properly using nasal speculum. Any swelling, deviation of septum, blockage should be observed. Nasal discharge may be evident. Inner surface of the mandible and inferior/palatine surface of the maxilla is inspected by opening the mouth widely (using proper light source). Teeth (missing, caries) should be numbered and labeled; ulcers; swelling from inner surface of the bone should be inspected. When swelling is present, its size, shape, extent should be observed. Nasopharynx should be examined. Epulis (swelling arising from gums), odontomes may be evident. Contour of the alveolus; alignment of teeth; trismus should be observed. Ears should be inspected using a speculum (**Figs 11.2A to 11.3**).



Fig. 11.2A



Fig. 11.2B

Figs 11.2A and B: Inspect the oral cavity and palate carefully. Use a spatula and light source.



Fig. 11.3: Inspect alveolus by properly retracting the lips.

Palpation

Palpation is done initially over the outer surface then inside by wearing a glove. Tenderness, swelling, fracture site (in mandible there is loss of continuity of lower border and crepitus) should be examined. Surface, consistency, tenderness, mobility, fixity should be ascertained while examining a swelling. Orbital margins of the maxilla should be palpated carefully on both sides for bone erosion, discontinuity, and swelling. Patient is asked to blow through one nostril while closing the other nostril. Free easier blowing means there is no nasal blockage. Only tenderness in the maxillary antrum suggests sepsis in the antrum. Area adjacent to loose teeth should be palpated. Entire alveolar margin of both upper and lower jaw should be palpated. Body, angle and lower part of the ramus of the mandible should be palpated from outside and inside. *Bidigital palpation* is done by placing one finger inside the mouth and fingers of other hand is placed outside to feel tenderness, irregularity, discrepancies, swelling, and thickening. It should be done on both sides for comparison (**Figs 11.4A to 11.8B**).

Movements of the Temporomandibular Joint

Joint can be felt by placing *little finger* in the external auditory canal with pulp facing *forward* and asking the patient to open and close the mouth (**Figs 11.9A to C**). Condylar movements cannot be felt in dislocated TM joint. *Dislocation* can be unilateral or bilateral. Partially opened jaw with deviation towards opposite side and hollowness behind the dislocated condyle can be felt. In bilateral dislocation mouth is opened and fixed (*prognathous deformity*). In normal opening of the jaw the distance is 2.5 cm between upper and lower incisor teeth. Joint movement is also checked by placing fingers over the *joint just below and in front* of the tragus. *Crepitus* due to osteoarthritis; click due to loose bodies can also be felt. *Ankylosis of TM joint* causes restricted mouth opening. Osteoarthritis, fibrosis of soft tissues around are the causes. It is often difficult to differentiate it from trismus. *Trismus* is due to muscular spasm (masseter and pterygoids) by inflammation (dental abscess, acute parotitis, partially erupted 3rd molar/wisdom tooth, pharyngeal, peritonsillar abscess); Risus sardonicus of tetanus; oral



Figs 11.4A to C: Eliciting the tenderness in maxilla and also checking the nasal blockage.



Figs 11.5A and B: Palpating inferior orbital margin for tenderness or disruption.

malignancy infiltrating the soft tissues beneath also causes restricted jaw opening. *Clicking* of jaw also occurs due to displacement of articular cartilage of the TM joint which is common in females. When the mouth is opened widely like in yawning the jaw gets suddenly locked with a snap in the ear; and patient cannot close the mouth later. Later each time mouth opening causes a click.

Sensation over the mental area, infraorbital region and other areas of trigeminal nerve should be checked when needed (**Figs 11.10A and B**).

Cervical lymph nodes should be palpated for significant enlargement (**Fig. 11.11**).



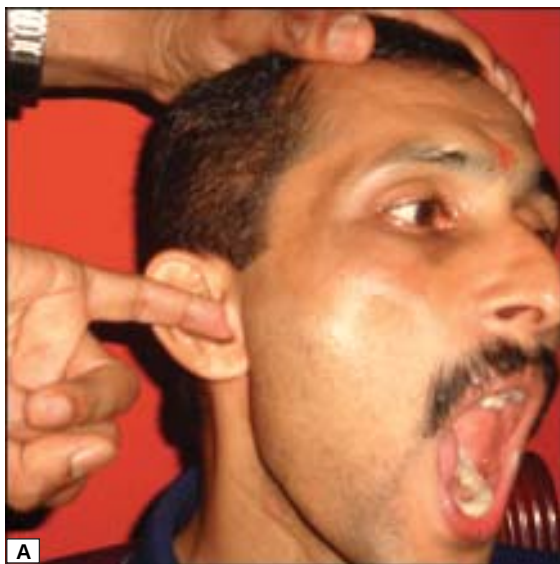
Figs 11.6A to C: Palpating the alveolar margins of the jaw and lower margin of the mandible.



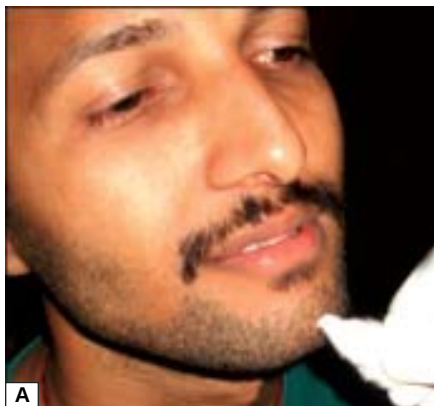
Fig. 11.7: Bidigital palpation of jaw (mandible) is important.



Figs 11.8A and B: Transillumination of maxilla is done by two methods. One is by illuminating the torch over the external surface of the maxilla in a dark room. Another is by placing the tip of illuminating torch into the mouth and mouth is closed in a dark room to see whether maxilla is transilluminating (normal) or not (pus or tumour).



Figs 11.9A to C: Temporomandibular joint movements should be checked both by placing little finger inside and from outside the ear.



Figs 11.10A and B: Sensation should be checked using cotton over the mentum in lower jaw; over the infraorbital region in upper jaw.



Fig. 11.11: Cervical lymph nodes should be palpated in jaw tumours.

Investigations for Jaw Disease

1. **Orthopantomogram (OPG):** It is a plain X-ray of the jaw and mandible which shows the entire mandible and partly maxilla in a single plane. It is better than X-ray mandible lateral view as it highlights proper dentition, inner and outer plates of mandible (**Figs 11.12A and B**). It is a rotational tomogram.

Indications: Jaw tumours—adamantinoma, dental cyst, dentigerous cyst, osteoclastoma; osteomyelitis of the mandible; fracture mandible; to see infiltration in carcinoma of oral cavity.

2. **CT scan** of jaw including neck and base of skull in maxillary diseases, tumours, trauma to assess extent. Sinus endoscopy.
3. **Biopsy**, discharge study, culture of discharge, FNAC of lymph node.

Maxillary Tumours

They are rare. Maxillary sinus is the *commonest site* of malignancy in paranasal sinuses. Ethmoids, and

sphenoids are next in order. It is common in people working in furniture industries, mustard gas industries, and leather industries. It is common in Bantus in South Africa where snuff with nickel and chromium is commonly used. Squamous cell carcinoma is the commonest type—80%. Adenocarcinoma, transitional cell carcinoma, salivary tumours, sarcomas, melanoma and Burkitt's lymphoma also can occur (**Fig. 11.13**).

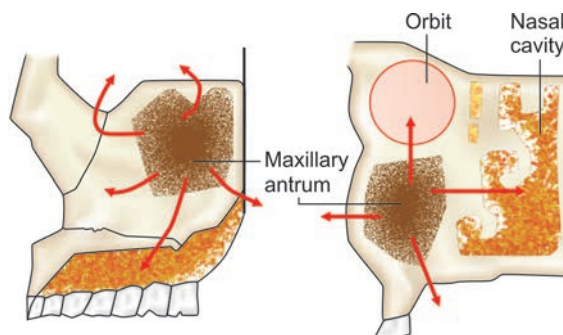


Fig. 11.13: Extensions of tumour from maxillary antrum.

Behaviour and Presentation

Initially may be asymptomatic or may present with *epistaxis* or *features of chronic sinusitis*. When it spreads to the floor, loosening of the teeth, necrosis, antro-oral fistula can occur. Extension medially causes nasal block, fungation, nasal discharge, blockage of nasolacrimal duct (epiphora). Extension anteriorly causes pain, anaesthesia and swelling in the cheek, ulceration and fungation in the skin of cheek. Spread above into the orbit causes epiphora, diplopia, proptosis. *Posterior spread is most dangerous* as it is not revealed easily. It causes postnasal discharge, pain, trismus, limitation of movement of temporomandibular joint. Involvement of upper deep cervical lymph nodes in later stage is common.

Differential diagnosis: Chronic sinusitis.

Classification

Ohngren's classification: An imaginary plane is drawn extending between medial canthus of eye and the angle of mandible. Growth situated above this plane is called as *suprastructural* which has got poor prognosis. Growth below this plane is called as *infrastructural* and has got better prognosis (**Fig. 11.14**).



Figs 11.12A and B: Orthopantomogram being taken and X-ray OPG look.

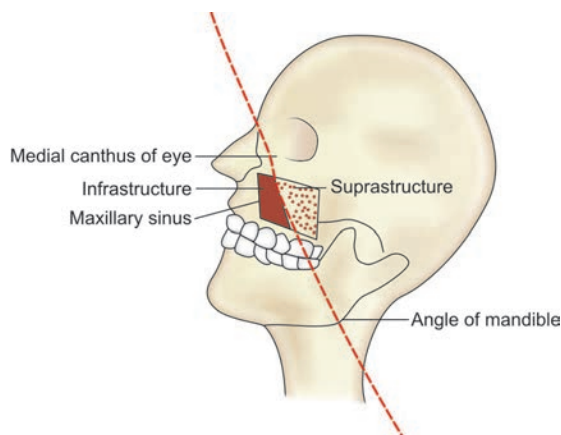


Fig. 11.14: An imaginary plane is drawn extending between medial canthus of eye and the angle of mandible and line in this plane is called as Ohngren's line.

Lederman's classification: Two horizontal lines are used, one passes through the floor of the orbit, another passes through the floor of the antra. These lines are called as *line of Sebileau*. (1) *Suprastructure type*: In this type olfactory area of nose, ethmoidal, sphenoid, and frontal sinuses are involved. (2) *Mesostructural type*: This involves maxillary sinus and nasal respiratory part. (3) *Infrastructural type*: This type involves alveolar process. Lederman's classification is further divided by two vertical lines over medial walls of the orbit to separate ethmoid sinuses and nasal fossa from maxillary sinuses (**Fig. 11.15**).

Diagnosis: X-ray of the part-opacity of the involved sinus with destruction of bony walls is seen. CT scan is ideal method (**Figs 11.16A and B**). Biopsy is done through nasal/oral route or on early stage through

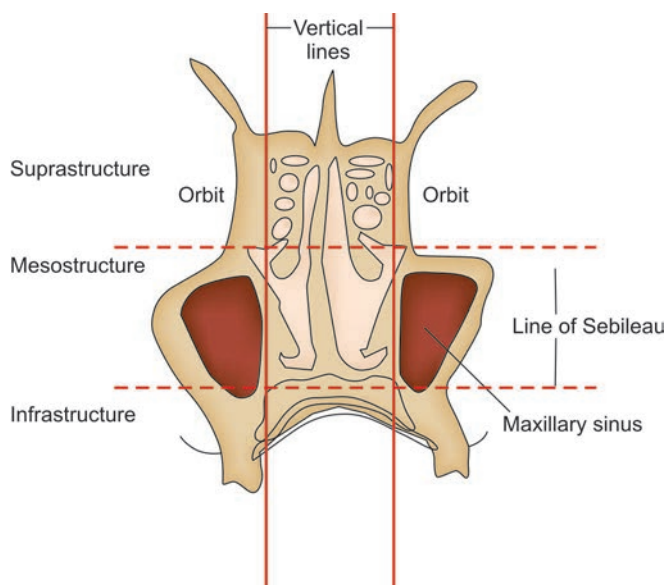
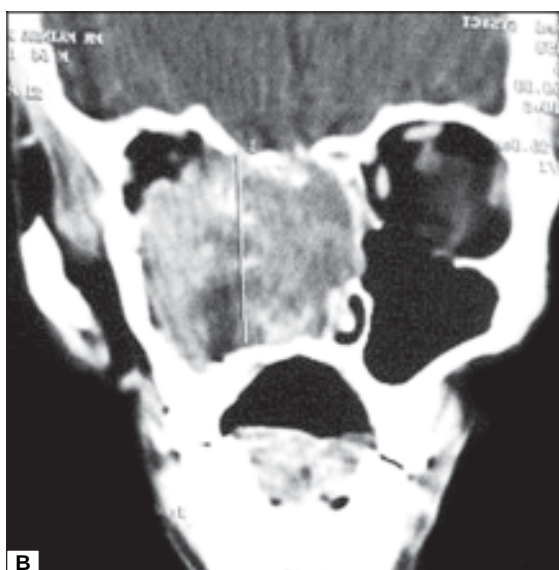


Fig. 11.15: Diagrammatic representation of Lederman classification.

TNM staging	Staging
T_1 Tumour limited to antral mucosa	Stage I $T_1 N_0 M_0$
T_2 Tumour causing bone erosion/destruction including extension into hard palate or middle meatus of nose.	Stage II $T_2 N_0 M_0$
T_3 Tumour invading bone of posterior wall of maxillary sinus/skin of cheek/medial wall of orbit/infratemporal fossa/pterygoid plates/ethmoid sinuses.	Stage III $T_3 N_0 M_0$
T_4 Tumour invading orbital contents beyond the floor or medial wall including orbital apex/cribriform plate/base of skull/nasopharynx/sphenoid or ethmoidal sinuses.	$T_1/T_2/T_3 N_1 M_0$
	Stage IVA $T_4 N_0 M_0, T_4 N_1 M_0$
	Stage IVB Any T $N_2 M_0$
	Any T $N_3 M_0$
	Stage IVC Any T Any N M_1



Figs 11.16A and B: Carcinoma maxillary antrum and CT picture.

Caldwell-Luc operation. Sinus endoscopy is done for detailed examination of sinus and for biopsy.

Epulis

Swelling arising from the gums is called as *Epulis* (gumboil, upon gum—Greek).

Congenital epulis: It is a benign condition seen in a newborn arising from gum pads. It is a variant of granular cell myoblastoma originating from gums. It is more common in *girls*. It is more common in *upper jaw*. It is not a malignant condition. It is well localised

swelling from the gum which is firm and bleeds on touch.

Fibrous epulis: It is a benign condition, can occur in any individual. It is the commonest type of epulis; it is firm or hard, commonly sessile rarely pedunculated slow growing benign fibroma arising from periodontal membrane. It is painless, well localised, hard/elastic, non-tender gray-pink swelling in the gum which bleeds on touch. It mimics squamous cell carcinoma. OPG; biopsy from the lesion is essential. Recurrence can occur if root is not removed properly.

Pregnancy epulis: It occurs in *pregnant women* due to *inflammatory gingivitis* usually during 3rd month of pregnancy. *Clinically* it resembles fibrous epulis or pyogenic granuloma. It usually resolves after delivery; otherwise it should be excised.

Granulomatous epulis: It is a mass of granulation tissue in the gum around a carious tooth. It is soft, bright red swelling which bleeds while brushing.

Myelomatous epulis: It is seen in *leukaemic* patients. Investigated for leukaemia by peripheral smear, bone marrow aspiration and biopsy.

Giant cell epulis: It is osteoclastoma causing ulceration and haemorrhage of gum. It is painless expanding swelling in mandibular part.

Carcinomatous epulis: It is squamous cell carcinoma of the alveolus and gum presenting as localised, hard, indurated swelling with ulceration.

Fibrosarcomatous epulis : It is fibrosarcoma arising from fibrous tissue of the gum. It is with variable consistency often softer, bluish red, progressive swelling which bleeds on touch.

Jaw Tumours

Classification

Swelling arising from the gums (Epulis): Congenital epulis; Fibrous epulis; Pregnancy epulis; Giant cell epulis; Myelomatous epulis; Sarcomatous epulis.

Swelling arising from the dental epithelium (Odontomes): **Benign odontogenic tumours:** *Epithelial*—Ameloblastoma; Calcifying odontogenic tumour; Odontogenic adenomatoid tumour; Enameloma; Composite odontoma, which may be either complex or

compound. It is odontogenic hamartoma containing all 4 layers—dentin, enamel, cementum and pulp. **Mesodermal tumour:** Odontogenic fibroma, myxoma; Cementoma, dentinoma. **Malignant odontogenic tumours:** Malignant ameloblastoma; Fibrosarcoma.

Cysts arising in relation to dental epithelium: Dental cyst; Dentigerous cyst.

Swelling arising from the mandible or maxilla: Osteoma and osteoblastoma; Torus palatinus and mandibularis; Fibrous dysplasia; Osteoclastoma; Osteosarcoma; Secondaries.

Surface tumours: Tumours from the surface which extend into the jaw (**Fig. 11.17**).



Fig. 11.17: Large jaw tumour – could be adamantinoma or osteoclastoma.

Ameloblastoma (Adamantinoma [Greek *Adamas* – strong/unconquerable], Eve's disease, Multilocular cystic disease of the jaw)

It arises from the dental epithelium probably from the enamel/dental lamina. It occurs commonly in mandible or maxilla. Occasionally it is seen in the base of the skull in relation to Rathke's pouch or in tibia. Histologically it is a variant of *basal cell carcinoma*. It is a *locally malignant tumour*. It neither spreads through lymph node nor through blood. Hence it is *curable*. It is usually unilateral. It can occur in a pre-existing dentigerous cyst. It is multilocular but can be unilocular.

Clinical features: Swelling in the jaw usually in the *mandible near the angle* extending to vertical ramus which attains a large size. It is gradually progressive, painless, smooth and hard with *intact inner table*. Outer table *expansion* is typical. Lymph nodes are not enlarged. It is common in males; common in 5th decade. It should be differentiated from osteoclastoma of mandible (here inner table is not intact); dentigerous cyst; dental abscess. Condition is curable by proper surgery. Recurrent adamantinoma can spread through blood. OPG shows eccentric expansion of the angle and vertical ramus of the mandible with trabeculations – *honeycomb* look (**Fig. 11.18**).

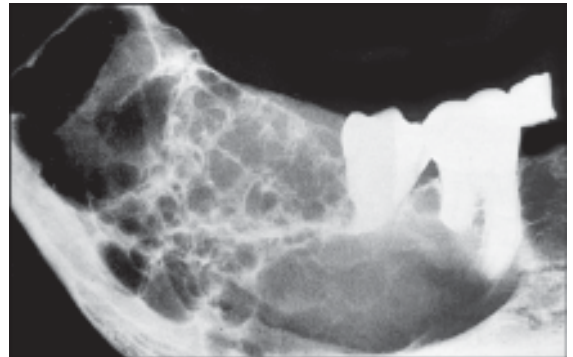


Fig. 11.18: X-ray showing adamantinoma with honeycomb look.

Curable malignancies

- Adamantinoma
- Basal cell carcinoma
- Verrucous carcinoma
- Papillary carcinoma thyroid
- Marjolin's ulcer
- Carcinoma colon

Dentigerous Cyst (Follicular Odontome)

It is a unilocular cystic swelling arising in relation to the dental epithelium from an unerupted tooth. It is common in lower jaw (in relation to premolar or canine), but can also occur in upper jaw; It occurs over the crown of unerupted tooth; commonly seen in relation to premolars or molars (**Fig. 11.19**). It causes expansion of outer table of the mandible; it is solitary and unilocular containing glairy fluid; histologically contains enamel derived squamous cells; common in younger age group; presents as painless swelling in the jaw which is smooth and hard; egg shell crackling



Fig. 11.19: Dentigerous cyst.

often may be present. It mimics dental cyst, adamantinoma, osteoclastoma. It can turn into adamantinoma. OPG shows well circumscribed translucent area in the jaw with permanent unerupted tooth within it (**Fig. 11.20**).

Dental Cyst (Radicular Cyst, Periapical Cyst)

It occurs under the root of a chronically infected dead erupted tooth. It is lined by squamous epithelium

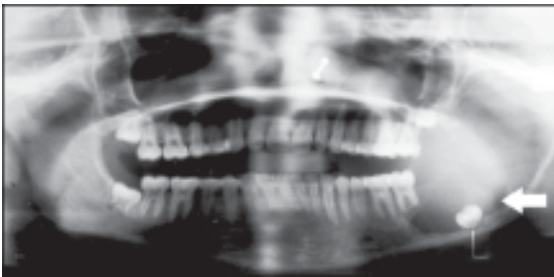


Fig. 11.20: Orthopantomogram showing dentigerous cyst.

derived by epithelial debris of *Mallassez*. It is an infective granuloma with epithelial proliferation which later gets degenerated to cause cystic fluid in the cavity. It is unilocular; contains mucoid material and cholesterol crystals; presents as a smooth, tender localized swelling in the jaw with expansion in relation to caries tooth; common in adult; common in maxilla adjacent to upper incisor or canine. OPG shows circular radiolucent area with clear often sclerosed margin in relation to the tooth of the maxilla. It can cause osteomyelitis of the mandible (**Fig. 11.21**).

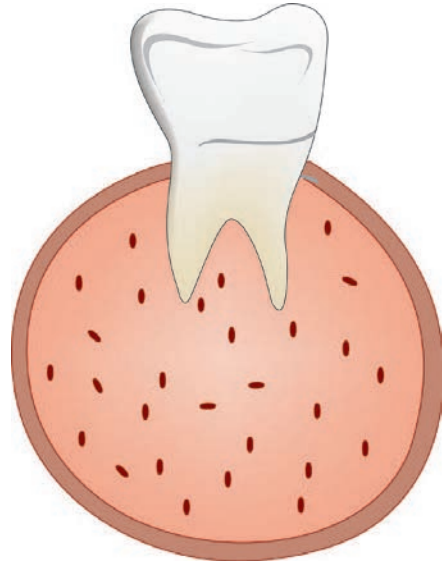


Fig. 11.21: Dental cyst.

Solitary Bone Cyst

It occurs in premolar or molar region of the mandible. Rounded cyst bulges outwards. Bone resorption with bone deposition in the margin is common. Fluid is yellowish with high bilirubin content. Haemorrhage with clot formation is known to occur.

Differences between Dental cyst and Dentigerous cyst:

	<i>Dental cyst</i>	<i>Dentigerous cyst</i>
Site	Maxilla – incisor / canine	Mandible – premolar / molar
Type of tooth	Erupted infected tooth often dead tooth	Unerupted permanent tooth
Location	Under the root of tooth	Over the crown of the tooth
Age	Adult	Younger age group
Complication	Osteomyelitis	Adamantinoma

Ossifying Fibroma

It occurs exclusively in jaw bones. It is common in young girls. In upper jaw it fills the maxillary antrum and later presents as well localised external swelling. Initial rapid growth ceases eventually and becomes stationary. *Alignment of teeth is known to occur.* X-ray shows soft tissue shadow with scattered bone deposition.

Fibrous Dysplasia

It is a self limiting disease where the medullary/spongy bone is replaced with fibroosseous tissue. It can be monoostotic or polyostotic. Monoostotic is common in long bones. In *polyostotic type* mandible also is commonly involved. Maxilla can be involved occasionally. It is *crab flesh white* in colour; containing islands of cartilages and cystic spaces. It can be bilateral in mandible (**Fig. 11.22A**). It presents as painless

swelling in the mandible of growing children; common in females; showing expansion of outer cortex but teeth are normal. Treatment is done only after cessation of skeletal growth. X-ray shows typical area of '*smoke screen translucency*' (**Fig. 11.22B**). Polyostotic fibrous dysplasia; pigmentation of the skin; precocious puberty in females is – *Albright's syndrome*.

Osteoclastoma (Giant Cell Tumour) of Mandible

It is giant cell tumour arising from epiphysis in young adults; common in long bones; can occur in mandible. It can be benign/intermediate or malignant (10%). Expanding swelling towards inner table of the mandible with cystic spaces; egg shell crackling; *discontinuity in inner table* are typical. Central part of the jaw either mandible (common) or maxilla is involved (in mandible body is commonly involved). When it is malignant spread can occur to lungs. Pathological fracture is known to occur. Displaced roots of adjacent teeth, loose teeth are common. Giant cells are due to fused spindle cells (not due to osteoclasts-misnomer). Giant cell epulis, brown tumour of hyperparathyroidism, dentigerous cyst and adamantinoma are differential diagnosis. X-ray and biopsy confirms the diagnosis.

Giant Cell Reparative Granuloma (Jaffe Tumour)

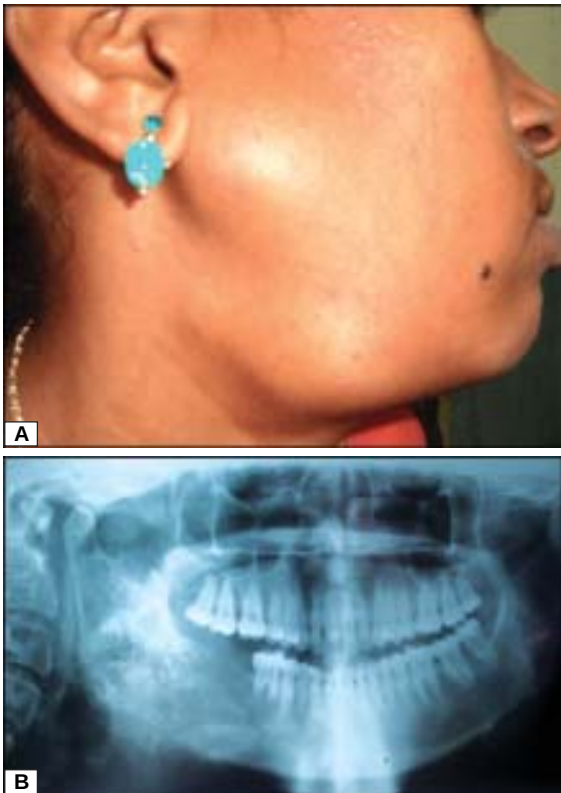
It is a swelling which occurs due to haemorrhage within the bone marrow. It contains vascular stroma, collagen and connective tissue cells. It is common in women. It causes painless enlargement of jaw. It can be treated by calcitonin (100 units/0.5 mg subcutaneously daily for 12 months) or surgical curettage.

Upper Jaw Tumours

Ivory osteoma, osteoclastoma, osteosarcoma, squamous cell carcinoma of maxillary antrum, carcinoma of hard palate are the examples (**Fig. 11.23**).

Lower Jaw Tumours

Fibrous dysplasia is common in mandible as it develops partly from membrane. Paget's disease of jaw, osteoclastoma, oral malignancy infiltrating the mandible are common types (**Fig. 11.24**).



Figs 11.22A and B: Fibrous dysplasia of mandible (A) and X-ray picture (B).



Fig. 11.23: Upper jaw tumour—from maxilla causing proptosis.



Fig. 11.24: Lower jaw tumour—could be adamantinoma mandible.

Burkitt's Lymphoma

It is *multifocal* childhood lymphoma common in Africa probably due to Epstein-Barr virus. It can occur in upper or lower jaw. It is common in premolar or molar area. When many areas are involved it occurs on same side of both maxilla and mandible. Disease expands outwards involving cheek and soft tissues outside. Lamina dura of teeth disappears. Neck nodes may get enlarged; retroperitoneal mass; hepatomegaly; ovarian tumour; renal, adrenal, pancreatic and mesenteric nodal involvement are common. Involvement of spinal nerves, salivary gland, breast, thyroid, bones, intracranial spread, and cranial nerve palsies are known to occur. Typical *starry sky pattern* in histology is obvious.

Alveolar Abscess (Dental Abscess)

It is due to spread of infection from root of the tooth into the periapical tissue. Initially it forms periapical abscess which later spreads through the cortical part of the bone into the soft tissues around forming an alveolar abscess. Disease begins in the pulp of tooth → pulpitis → spread to root → localised osteitis → abscess formation → spread into soft tissues outside in cheek → initially diffused later localised swelling in the jaw with redness and oedema of gum. Initial dull continuous pain later becomes severe excruciating pain. Fever, trismus, often dysphagia, palpable tender neck lymph nodes occur eventually. Oedema, pain in the floor of the mouth may occur (**Fig. 11.25**). Swelling may burst spontaneously to form a sinus outside.



Fig. 11.25: Typical dental abscess.

Bacteria: Staphylococci, streptococci, anaerobic bacteria and gram-negative organisms. Alveolar abscess occurring in relation to upper lateral incisor will not present outside but produces swelling on the palate medially. Abscess in relation to wisdom tooth also opens medially (not outside) and may cause Ludwig's angina. X-ray will reveal rarefaction of the root of the tooth only after 10 days.

Complications: Septicaemia; spread of infection into other spaces like parapharyngeal spaces; sublingual and submandibular spaces causing Ludwig's angina; oedema of epiglottis and respiratory distress; spread to pterygoid space and along pterygoid muscles through emissary vein → cavernous sinus thrombosis; upper canine tooth abscess → medial corner of eye → angular vein thrombophlebitis → cavernous sinus thrombosis; submasseteric abscess; lower incisor abscess can cause abscess in the chin and later median mental sinus; chronic osteomyelitis of the jaw with discharging sinuses.

Osteomyelitis of Jaw

It can be in the maxilla or mandible.

Causes: Alveolar abscess leading into osteomyelitis; recurrent dental infection; trauma; after dental extraction; surgeries of the jaw; postradiotherapy osteomyelitis (osteoradionecrosis).

Types: *Acute:* Common in children; maxilla or mandible may get involved (**Fig. 11.26**); swelling, redness, fullness is the features; pus may trickle through nostril if it is in maxilla. *Subacute:* It is the commonest type; common in adult; apical sepsis, endarteritis, bone necrosis is the pathology; common in mandible; rare in maxilla due to existing network vasculature which prevents endarteritis. Compression over inferior dental nerve causes numbness in chin in distribution area of mental nerve. Pain, swelling, tenderness, irregularity, thickening are typical. *Chronic:* It is also common in mandible; apical abscess, alveolar abscess, trauma, radiation, chemical like phosphorus, tuberculosis, syphilis, actinomycosis are the causes. Pain, thickening, irregularity, discharging sinus, sequestrum in the discharge, discomfort are the features. Infection from lower incisor causes median mental sinus. X-ray shows features of osteomyelitis with new bone formation and sequestrum.



Fig. 11.26: Osteomyelitis of mandible.

Actinomycosis: Faciocervical is the commonest type; lower jaw is commonly involved; infection begins at carious tooth; indurated gums → nodules → abscess → multiple sinuses → discharging sulphur granules with normal X-ray (*Ray fungus*) → *Actinomycosis israelii* is the causative agent.

Cherubism (Cherub–Angelic Being)

It is an autosomal dominant familial condition occurring in first year of life. It shows giant cell granuloma with fibrous tissues in the jaw. It is commonly *bilateral*; commonly seen in *angles of the mandible* and also in maxilla. It presents as diffuse enlargement of maxilla and both sides of the mandible; bulging of the cheek causes pull of the lower eyelid. Hence child appears like, as if looking upwards (*winged face of angelic babies*); interference of the development and eruption of the teeth. It is a self limiting disease. Often requires dental care and treatment for proper dentition.

Treacher-Collins Syndrome

It consists of mandibulofacial dystosis; hypoplasia of the zygomatic bone and mandible; antimongoloid slant to the palpebral fissure; coloboma of lower eyelid; Low ear lobule with deficient middle ears. It is familial.

Pierre-Robin Syndrome

It is a congenital entity consisting of – cleft palate alone; mandibular hypoplasia; cyanotic episodes, deficiency in transforming growth factor; defective

sucking and tongue falling backwards in infants, cryptorchidism.

Micrognathism and Prognathism

Excessively small mandible is called as *micrognathism*. Backward displacement of tongue in neonates with micrognathism can cause respiratory distress. Here oral cavity is small. *Prognathism* is where mandible is larger than average with protrusion. Occasionally maxilla is hypoplastic.

Diseases of the Palate

Cleft palate; *Torus palatinus*—a bony hard swelling in the centre of the hard palate; nasopalatine cysts; *Epstein's pearls* at the junction of soft and hard palates in the midline in infants due to retained developmental cell rests; apical cyst or abscess; minor salivary gland tumour—commonest site is palate; Maxillary tumour extending into the palate; Squamous cell carcinoma of the palate; gummatous perforation in the middle of the palate seen in congenital syphilis; perforation of the palate anywhere in carcinoma palate.

Nasopalatine Cyst

It develops in incisive canal; causes spherical bone cavity behind upper incisors; composed of epithelial lining with fibrous capsule with mucous secreting cells. It occurs in the midline of palate.

Maxillofacial Injuries

It may be due to road traffic accidents, assaults, bullet injuries or sport injuries.

Classification

Fracture in maxillofacial region can be grouped as: Fracture lower third that comprises mandible; Fracture middle third that comprises maxilla, zygoma and nose; Fracture upper third of the face involving part of the orbit, frontal bones.

Maxillofacial fracture also can be grouped as: Fracture of the face which do not involve the dental occlusion; fractures of zygoma and nose; fracture which involves the dental occlusion; fracture mandible and maxilla.

Soft-tissue injuries: Lacerations, contusions, cut wounds, etc; Eyelid injuries with black eyes; Facial nerve injury; parotid duct injury; lacrimal apparatus injury.

Injuries to the facial bones: *Fracture nose*—Patient presents with pain and swelling in the nose with deviation and displacement. Injuries to the *maxilla*; *zygomatic* bone injuries; *mandibular* bone fracture and mandibular dislocation; *orbital bone fracture*: presents with diplopia, enophthalmos, infraorbital nerve sensory loss.

Respiratory Obstruction

Causes: Oronasal airway block can occur by blood, clot, vomitus, foreign body, dentures, teeth, saliva, bone pieces, etc; Backward falling of tongue can cause obstruction of the nasopharynx and oropharynx. It is common in bilateral mandibular fracture; occlusion of the nasopharynx and oropharynx can occur in fracture maxilla with posterior and inferior displacement; haematoma in floor of the mouth or posterior oral cavity can cause airway block; other features include oedema of larynx/tongue/posterior third of oral cavity/pharynx; surgical emphysema.

Haemorrhage in Maxillofacial Injuries

Haemorrhage in maxillofacial injuries is usually not life threatening. But it should be identified and controlled properly. In association with other internal injury, such haemorrhage may be important to cause the circulatory failure. Haemorrhage may be due to—Soft tissue bleeding; bleeding from inferior alveolar artery, palatine vessels; nasal bleeding.

Clinical Features

Localised swelling due to haematoma; facial oedema; bleeding with open wounds; asymmetry which is clinically confirmed by observing supraorbital ridges, nasal bridge; localised tenderness; step deformity; trismus; diplopia; features of associated injuries like intracranial, abdominal or thoracic injuries.

Investigations: X-ray face; CT scan of head/jaw.

General treatment for faciomaxillary injuries: Suturing of soft tissues; Airway maintenance; Control

of bleeding; Pain relief; Control of infection; Treating the individual fractures (**Fig. 11.27**).

Fracture Middle Third Area

It includes—maxillae, zygomatic bones, palatine bones, nasal bones, lacrimal bones, inferior conchae (one on each side), the vomer, ethmoid and its attached conchae, pterygoid plates of sphenoid.

Zygomatic Complex Fracture

Classification

Simple fracture which is stable and undisplaced—
Here fracture line passes across the infraorbital foramen downwards over anterior wall of the antrum.

Simple fracture which is displaced medially. It may be associated with rotation/tilt in vertical axis, either

Fracture middle third includes fracture maxilla, zygoma and nasal bones.

Fractures not involving occlusion	Fractures involving occlusion
Central • Fracture nasal bones and or nasal septum • Fracture of frontal process of maxilla • Fractures of above two extending into ethmoid- nasoethmoid • Fractures above three which extends into frontal bone – fronto-orbito-nasal dislocation Lateral • Fractures involving zygomatic bone, arch and maxilla excluding the dentoalveolar component	Dentoalveolar Subzygomatic • Le Fort I-low level either unilateral or bilateral • Le Fort II-pyramidal either unilateral or bilateral Suprazygomatic Le Fort III –high level • Craniofacial disjunction—unilateral or bilateral

Le Fort classification (Fig. 11.28)

(Rene Le Fort – French surgeon classified these fractures by dropping rocks on the face of the cadavers and later dissecting the area for study and research and published paper in 1911)

Types	Features
Le Fort I (Guerin's fracture-low level) (floating fracture, horizontal fracture of maxilla) • It runs horizontally above the floor of the nasal cavity involving lower third septum, palate, alveolar process of maxilla and lower third of pterygoid plates of maxilla	• Bleeding from nose • Posterior gagging of occlusion • Upper lip swelling • Palatal echymosis • Occlusion derangement • Floating maxilla
Le Fort II (pyramidal fracture) • From the nasal bones at topmost, fracture runs laterally towards lacrimal bones, medial wall of orbit, infraorbital margin, through or medial to infraorbital foramen and backwards below the zygomaticomaxillary area through lateral wall of maxillary sinus and pterygoid plates. Zygoma is intact with skull base	• Oedema of middle third face • Both sides circumorbital and subconjunctival ecchymoses • Nasal bleeding/obstruction/deformity • Deformity of face (dish face), diplopia • Retroposition of maxilla with posterior gagging • Limitation of ocular movements, CSF rhinorrhoea • Tenderness and separation of infraorbital margin
Le Fort III (craniofacial disjunction, high level) • Here fracture runs parallel to skull base. It passes through the nasal bone, lacrimal bone, ethmoid bone, optic foramen, inferior orbital fissure, pterygomaxillary fissure and lateral orbital wall with frontozygomatic suture with zygomatic arch	• Lengthening of face • Enophthalmos, ocular level depression • Hooding of eyes, occlusal plane tilting • Entire facial skeleton moves as a single block • Tenderness and separation of suture line • Diplopia • Trismus, teeth mal-alignment <i>Guerin sign:</i> Haematoma at greater palatine foramen



Fig. 11.27: Maxillary fracture

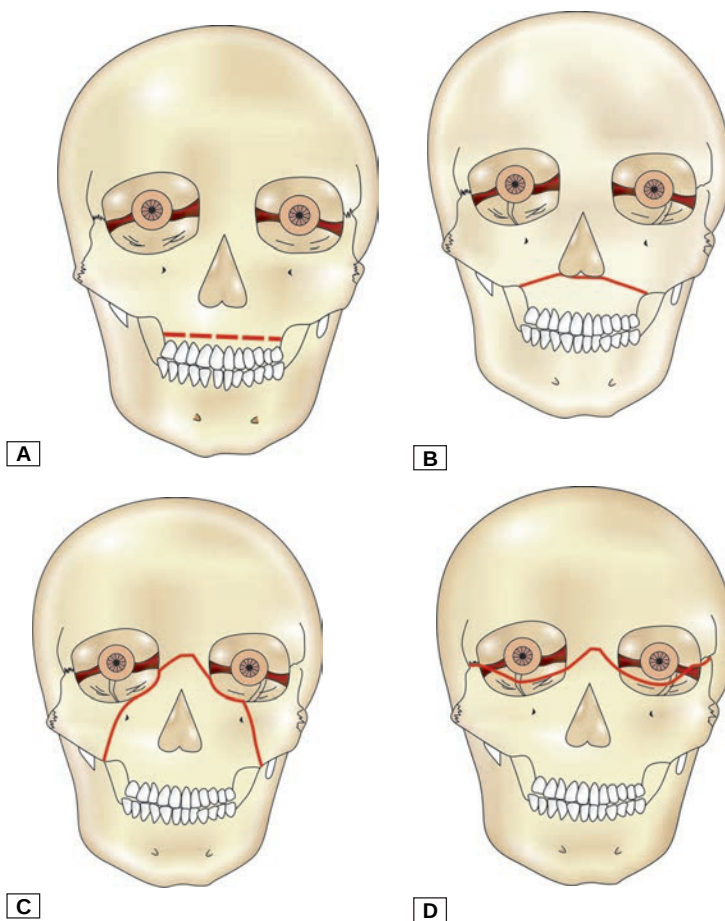
medial tilt or lateral tilt. Infraorbital nerve may get compressed or branches of superior dental nerve may get torn.

Unstable fracture with rotation around horizontal axis with medial tilt or lateral tilt.

Comminuted fracture extending into the floor of the orbit.

Fracture of the zygomatic arch causes a localised depression of the arch which displaces medially and tends to impinge on the coronoid process of the mandible.

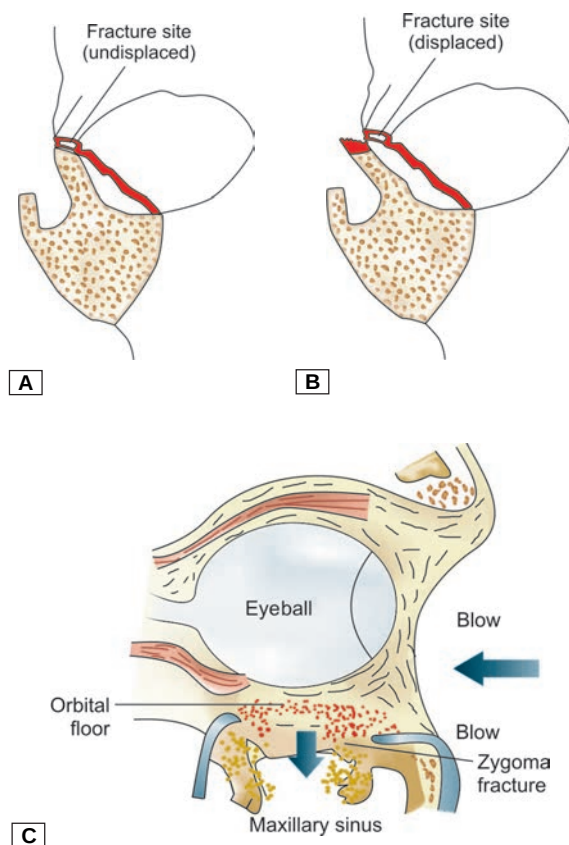
'Blow-out' fracture of the orbit is due to direct blunt trauma on the eyeball causing depressed comminuted



Figs 11.28A to D: Le Fort classification—different types and also dentoalveolar fracture. A—dentoalveolar; B—Le Fort I; C—Le Fort II; D—Le Fort III (Refer Table on Pg. 270 for details).

fracture of the orbital floor with herniation of the orbital fat into the antrum.

Enbloc dislocation of zygomatic bone medially/inferiorly/postero-laterally (**Figs 11.29A to C**).



Figs 11.29A to C: Diagrams showing different types of zygomatic fractures.

Clinical Features

Swelling and bruising in the cheek with sub conjunctival haemorrhage; Flattening of the cheek prominence; Step in the margin of the bony orbit at the infraorbital foramen; Sensory loss over the supply of the branches of the superior orbital nerve—teeth on the affected area are anaesthetic on percussion; Sensory loss over the supply of the infraorbital nerve usually over infraorbital region, upper lip and alar region of the nose – common; Enophthalmos is due to herniation of the orbital fat across the fracture floor of the orbit

into the antrum; Diplopia is due to entrapment of the inferior rectus muscle preventing upward rotation of the eyeball while looking up; Trismus with marked restriction of the lateral movements; Epistaxis, lowering of pupil level; Infraorbital ecchymosis of the orbit is called as *Panda sign*.

Investigations—30° occipitontental X-ray is commonly used but often obliquity of X-ray may be increased to 60°. In X-ray, findings observed are—Fracture line near infraorbital foramen, zygomatic arch and lateral wall of the antrum; Orbital floor line for fracture; Opacity in the antrum due to blood. CT scan is done to see orbital depression and herniation of orbital fat.

Fracture of the Mandible

Types (Fig. 11.30)

- I. At the *neck of the condyle* as it is the weakest point. The condyle is displaced in front and medially often with dislocation. Painful jaw movement is the clinical feature. It may be unilateral or bilateral.
- II. At the *angle of the mandible*: If fracture is upwards and inwards, it is impacted and undisplaced. So it is a favourable fracture. If fracture is downwards and outwards, it gets displaced and so it is an unfavourable fracture. It needs open reduction using wires.

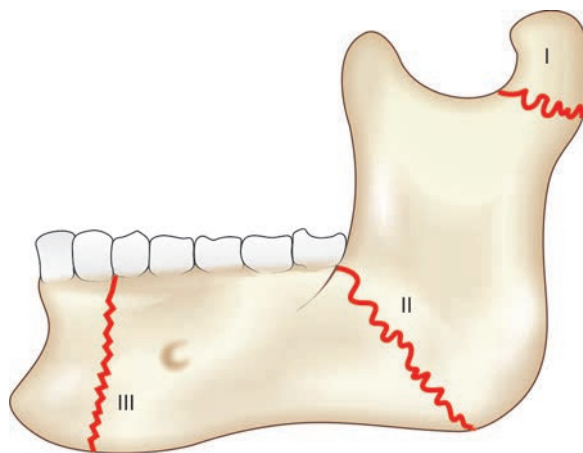


Fig. 11.30: Types of mandibular fractures.

Other classifications

Classification of the fracture mandible	
Depends on the type	Depending on the anatomical site (Fig. 11.31)
• Simple • Compound • Comminuted • Pathological • Green stick fracture in children	• Dentoalveolar fracture • Condylar fracture • Coronoid fracture • Fracture ramus of the mandible • Fracture angle of the mandible • Fracture in the body of the mandible • Symphyseal region fracture

Dentoalveolar fracture	
Features	Management
• Horizontal fracture below the alveolar margin • Dentoalveolar segment will be freely mobile • Tooth may get split vertically / horizontally • Derangement in occlusion and alignment • Gingival laceration • Bleeding • Infection and late osteomyelitis of mandible	• Look for other injuries in face • X-ray face to see injuries • Dentoalveolar segment reduction and placing jaws in central occlusion position • Stabilization using interdental wires or arch bars • Liquid diet for 3-4 weeks

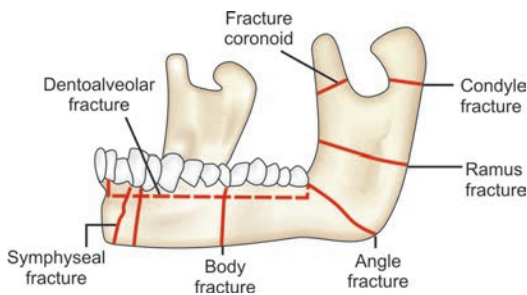


Fig. 11.31: Different sites of fracture mandible.

III. Fracture near the mental foramen through the canine fossa. This fracture causes displacement. Such bilateral fractures can cause pull on digastric and geniohyoid muscles precipitating fall of tongue backwards which will block the airway.

Guardsman fracture is direct fracture of symphysis and indirect fractures of both the condyles of the mandible. In olden days guards of queen who are in attention position used to faint and fall forward to get these fractures.

Clinical Features

Pain and tenderness in the lower jaw with bruising over the surface. Haematoma in the floor of the mouth

is called as *Coleman's sign*. Difficulty in opening the mouth, speech and swallowing; anaesthesia of the lower lip due to compression of inferior dental nerve; deranged dental occlusion; step deformity; blood stained saliva is common as often mandibular fracture is of compound type because mucoperiosteum is adherent to mandible (Fig. 11.32).

Complications of fracture mandible: Obstruction of the airway; Osteomyelitis of the mandible; Trismus; Speech disturbances.

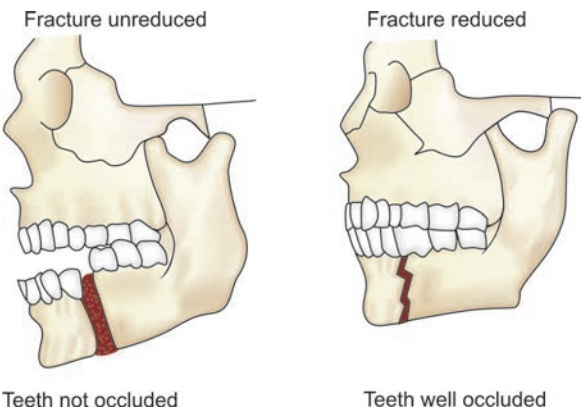


Fig. 11.32: Unreduced and reduced fractures of the mandible.

12

Examination of Salivary Gland

History

Swelling: Duration—progress, recent increase in size; Pain, whether initially painless now has become painful. Swelling of short duration with pain, trismus could be due to acute parotitis. Often it is bilateral in children due to viral cause (mumps). Bilateral enlargement of parotid along with other salivary glands and lacrimal gland is called as *Mikulicz syndrome*. Excessive salivation, joint pain along with enlargement of all salivary glands is called as *Sjogren syndrome*. Pleomorphic adenoma is slow growing tumour of long duration. Recent increase in size of swelling is important which suggests malignant transformation probably from a pre-existing pleomorphic adenoma. Adenolymphoma is slow growing tumour from lower pole of the parotid (**Fig. 12.1**).



Fig. 12.1: Typical parotid swelling—pleomorphic adenoma.

Pain: Duration/type/severity/radiation should be asked. Sudden onset of severe pain is a feature of acute parotitis. Throbbing excruciating pain may be a feature of parotid abscess. Colicky pain during meals is a feature of salivary calculus with sialadenitis. Stone is more common in submandibular salivary gland but can also occur in parotid gland.

Fever is a feature of acute sialadenitis or abscess. Acute sialadenitis with suppuration is common in parotid. Parotid abscess is usually unilateral. Mumps in children is bilateral. Neoplastic condition once necrosed can cause fever.

Difficulty in opening mouth can occur in acute parotitis, submandibular sialadenitis, and malignancy extending into the soft tissues.

History of excess salivation during meals/more pain during meals/swelling becoming more prominent during meals should be asked. It is a feature of stone in the salivary duct. Presence of sinus, its formation, discharge, etc. should be asked. Discharge from sinus/fistula is usually saliva. Its quantity, duration, colour whether increases while taking food should be clarified.

Recent increase in size suggests malignant transformation.

History of impairment of function like drooling of saliva, inability to close eyes, tears in the eye, asymmetry of face, difficulty in opening of the mouth should be asked.

History suggestive of metastases in case of malignant salivary tumours like of lungs, bone, brain should be asked.

Past history of surgery for parotid or submandibular swellings should be asked. Recurrent parotid tumours are known to occur in pleomorphic adenoma and malignancies. Detailed history of surgery, its nature of biopsy, postoperative management should be taken.

Past history of radiotherapy in head and neck region; past history of other malignancy in the body.

Personal history of alcohol intake is relevant in bilateral parotid enlargement.

Local Examination

Inspection

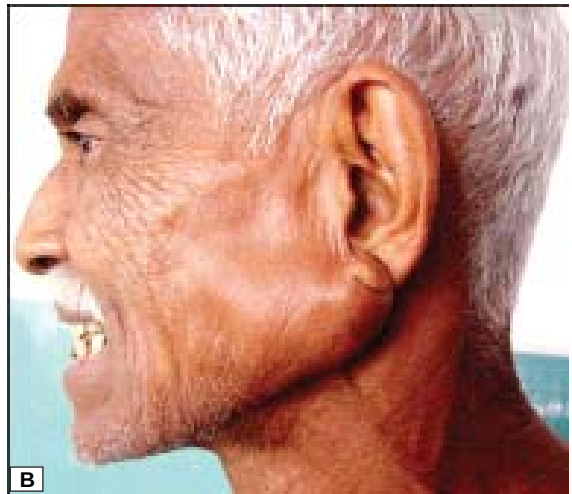
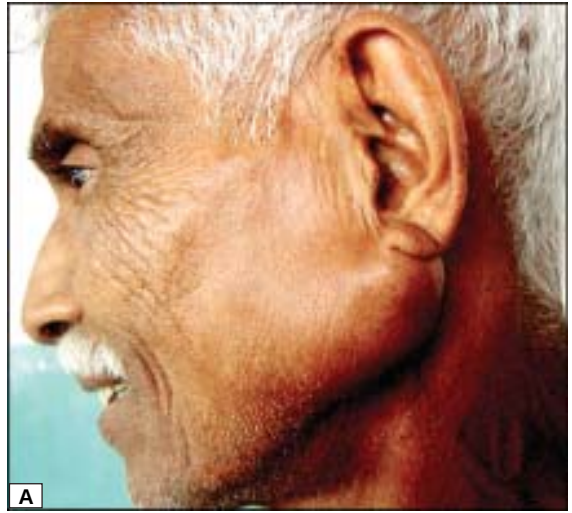
Swelling is examined in detail. Position of the swelling is noted. Parotid swelling is below, behind and in front of the ear lobule. Parotid enlargement shows typically raise in ear lobule. Normal hollow/depression just below the ear lobule are obliterated. Size, shape, extent, skin over the swelling should be inspected (**Figs 12.2 and 12.3A and B**). Skin is red and oedematous in parotid abscess or inflammatory conditions. In salivary calculus (submandibular calculus), swelling immediately becomes more prominent when lemon juice or chocolates are given to the patient to drink or eat.

Deep lobe of parotid enlargement is checked by inspecting the oral cavity for any bulge in the tonsil and lateral wall of pharynx. Floor of the mouth should be inspected for enlargement of deep lobe of the submandibular salivary gland (**Figs 12.4A to 12.5C**).

Stenson's parotid duct should be inspected opposite to 2nd upper molar tooth (**Figs 12.6A and B**). Cheek should be retraced using spatula and light source to inspect the duct properly. In suppurative parotitis pus



Fig. 12.2: Inspection of parotid should be done carefully by holding the ear lobule.



Figs 12.3A and B: Typical parotid swelling with ear lobe raised. Facial nerve should be tested by clenching the teeth.

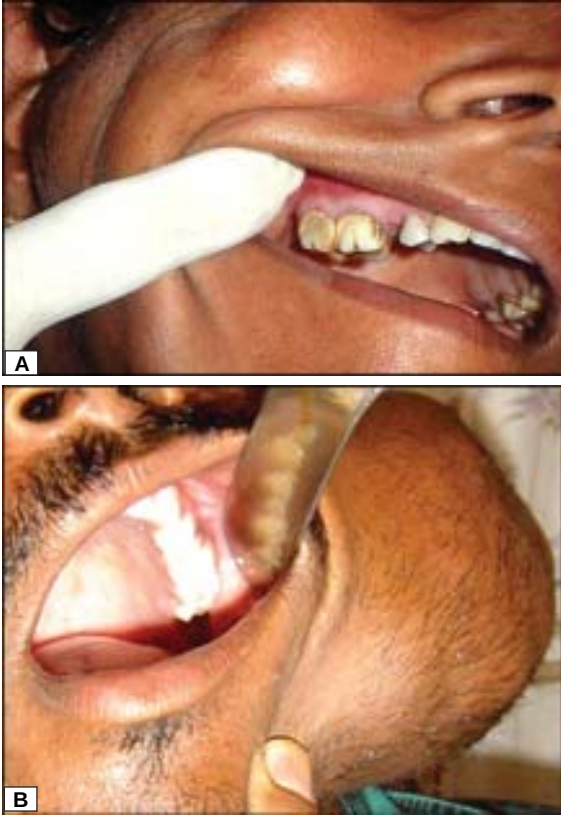
may be seen gushing out of the duct orifice after gentle pressure over the parotid gland. Blood in the duct orifice (ampulla of duct) may be due to malignant parotid tumour. Opening and course of the submandibular salivary duct (Wharton's) should be inspected after raising the tip of the tongue over the palate. Duct orifice may be inflamed and oedematous with discharging pus from it. Often stone may be visible in the duct orifice. Orifice is situated on either side of the frenum linguae. Duct with impacted stone blocks the salivary flow and hence that side orifice looks dry whereas normal side orifice looks wet due to normal



Figs 12.4A and B: Deep lobe of the parotid should be inspected from inside.



Figs 12.5A to C: Inspection of submandibular salivary gland swelling. Oral cavity is also should be inspected.



Figs 12.6A and B: Stenson's parotid duct should be examined opposite 2nd upper molar.



Fig. 12.7: In submandibular salivary gland enlargement of Wharton's duct should be inspected by raising the tip of the tongue over to palate.

Palpation

Swelling should be palpated like for any other swelling—local rise of temperature (**Fig. 12.8**) tenderness; surface; consistency; mobility; *curtain sign*; skin is free or not; extension to deeper plane; relation to masseter and mandible. *Curtain sign* is—deep fascia/parotid sheath is attached above to the zygomatic bone and so swelling arising from parotid gland cannot be moved up beyond zygomatic bone wherein deep fascia acts like a curtain to prevent its further mobility.

salivary flow (**Fig. 12.7**). Two small dry swabs are placed over the orifices on each side and patient is asked to take stimulant like lemon juice; after a minute swabs are taken out and inspected; swab on the side with impacted orifice will be dry; swab over normal orifice will be wet.

Skin over the swelling should be inspected. Redness suggests sialadenitis. Sialadenitis is inflammation of the salivary gland. Ulceration or fungation may develop in advanced carcinoma parotid. Sinus or salivary *fistula* should be inspected for discharge and location. If parotid fistula is in masseteric relation then it is from the gland; it will be premasseteric if it is from the duct.

Inspection of neck region for enlarged cervical nodes is also done.



Fig. 12.8: Temperature over the surface should be checked.

Swelling superficial to deep fascia can be moved beyond the level of the zygomatic bone above. Initially mobility of the swelling is checked in both directions; then patient is asked to clinch the teeth so that *masseter gets contracted* and mobility is checked again (**Figs 12.9 and 12.11A and B**). If mobility is restricted then swelling is adherent to masseter muscle. Nonmobile swelling means it is adherent to bone beneath. Whether skin is adherent to swelling or not should be checked (**Figs 12.10A and B**). *Consistency* is variable in different conditions—pleomorphic adenoma is firm but can be hard with smooth surface. Malignant swellings often have nodular surface and hard consistency; adenolymphoma (Warthin's) is smooth, soft often fluctuant and usually not transilluminant. *Tenderness* suggests that it could be abscess, necrosis in a tumour or deeper infiltration. Scar, fistula on the surface should be palpated.



Fig. 12.9: Lower margin of the enlarged parotid gland should be assessed.

Submandibular salivary gland swelling also should be examined similarly like any other swelling. Its medial, posterior extension, relation of the swelling to the lower margin of the body of the mandible should be checked (**Figs 12.12A to D**). Submandibular salivary gland enlargement occurs as a result of chronic sialadenitis or neoplastic conditions. Its surface is usually smooth whereas submandibular lymph node enlargement is usually nodular. Best way of palpating the *submandibular salivary gland* is by *bidigital palpation* (**Fig. 12.13**). It confirms swelling as



Figs 12.10A and B: Checking the mobility and skin fixation of the parotid swelling.

submandibular salivary gland and also deep lobe and duct can be palpated. Duct is palpated from behind forwards. First dentures if present should be removed. Index finger of one hand is placed over the floor of the mouth medial to alveolus and lateral to tongue pushing the finger as deep as possible; fingers of other hand are placed outside under the mandibular margin to push the swelling upwards. By this way the finger inside the oral cavity not only helps to feel the deep lobe of the salivary gland which is deep to mylohyoid muscle; but also the superficial lobe and often duct



Figs 12.11A and B: Mobility of parotid swelling should be checked in two directions.



Figs 12.12A to D: Submandibular salivary gland palpation – skin fixity (pinching); extent, margin and mobility. Fluctuation should be elicited in a soft swelling.



Fig. 12.13: Submandibular salivary gland bidigital palpation.

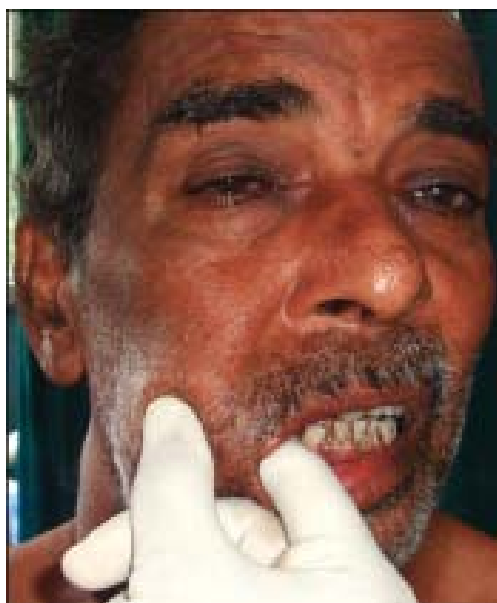


Fig. 12.14: Parotid duct palpation.

can be better assessed by this method. *Submandibular lymph node is not bidigitally palpable* as it is outside the mylohyoid muscle, superficial to the gland. Stone in the Wharton's duct also can be palpated by this method. Shape, size, consistency can be assessed by this method.

Intra oral examination: Parotid duct should be palpated using one finger inside the cheek and thumb outside the cheek (**Fig. 12.14**). Duct can be better felt when masseter is taut. Only anterior part of the duct is felt. Enlarged *deep lobe of the parotid* can

be felt by *bidigital palpation* with index finger of one hand placed inside the mouth in front of the tonsil and behind the 3rd molar tooth and fingers of the other hand placed outside behind the ramus of the mandible (**Figs 12.15A to 12.17**).

Features of parotid swelling

- Ear lobule raise
- Swelling in parotid region
- Swelling occupying the groove between posterior part of the mandible and mastoid process
- Moves upwards upto zygomatic bone—*curtain sign*



Figs 12.15A and B: Palpation of deep lobe of the parotid gland.



Fig. 12.16: Submandibular salivary gland duct should be palpated per orally.



Fig. 12.17: Mandible should be palpated bidigitally for relation of tumour, thickening, and tenderness.

Palpation

Tenderness / temperature / extent / size / surface / consistency / mobility / fixity / plane of the swelling / masseter involvement / facial nerve involvement / skin over the swelling.

Parotid duct palpation—by rolling the finger across the masseter muscle while patient is clenching the teeth to make masseter taut. Terminal part of the duct is palpated bidigitally using index finger inside and thumb outside. Palpation of oral cavity / bidigital examination for deep lobe is done with one finger inside the mouth behind the tonsillar fossa and the other outside in parotid region. All features of facial nerve palsy—inability to close eye / difficulty in blowing / altered nasolabial groove / clinching of teeth.

Neck nodes should be examined.

Examination of other salivary glands should be done. Relevant findings should be elicited in case of submandibular salivary gland enlargement.

Differential Diagnosis for Parotid Enlargement

Idiopathic hypertrophy of masseter muscle: It is a rare entity but presents like a swelling. When teeth are clenched entire swelling hardens; but when relaxed swelling softens. It often can be bilateral.

Preauricular lymph node enlargement: Swelling lies in front of the tragus; normal depression below and in front of the ear lobule is not obliterated; it may be suppuration, adenitis, tuberculosis or lymphoma. It feels more superficial.

Rarely parotid and paraparotid/subparotid lymph nodes may be enlarged as secondaries from primary oral mucosa and skin malignancies of head and neck region (but these things are very rare and so students should not consider in usual clinical practice unless it is relevant). Still rarely parotid gland may be enlarged as nonmetastatic obstruction of the duct by carcinoma cheek.

Differential Diagnosis for Submandibular Salivary Gland Enlargement

Enlarged submandibular lymph nodes: Bidigital palpation helps to confirm it. Lymph nodes are not bidigitally palpable.

Enlarged facial lymph node lies adjacent to facial artery at the lower margin of the mandible which can be moved above the level of the margin of the mandible into the face.

Functions of facial nerve should be checked. It is involved in malignant growth where nerve is infiltrated. It is involved early in adenoid cystic carcinoma; *carcinoma ex pleomorphic adenoma*. It is involved late in mucoepidermoid carcinoma.

Patient finds *difficulty in closing eyes* (orbicularis oculi); eye contains tear which does not fall; *difficulty in chewing food* (buccinator); *difficulty in talking, laughing, blowing, and whistling* (orbicularis oris).

Upper face:

Orbicularis oculi: Patient may not be able to open his eyes. In facial nerve paralysis eyes can be easily opened by the examiner when patient closes his eyes tightly.

Frontal belly of occipitofrontalis: Absence of furrowing in the forehead while looking upwards.

Corrugator supercilii: Absence of corrugation in the forehead while frowning.

Lower face:

Buccinator: While blowing with mouth closed, tone can be felt in the cheek.

Orbicularis oris: Inability to whistle.

Levator anguli oris: Deviation of angle of mouth towards opposite side while showing teeth.

Platysma: Loss of normal contraction while stretching the neck.

In supranuclear (upper motor neuron lesion) paralysis upper face escapes due to bilateral cortical representation.

Taste sensation and general sensation (lingual nerve) should be checked. Patient is not allowed to speak but asked to write in a paper. Taste material is instilled on the surface of the diseased side first and then normal side. Prior to each instillation patient should wash his mouth with warm water. Usually four substances are used. After 10 seconds patient should identify the substance and write. *Facial nerve serves 3 tastes*—*salt* (rock salt) on the tip of tongue; *sweet* using sugar syrup on the tip of the tongue; *sour* using lemon juice on the lateral aspect of the tongue. *Bitter taste* is mediated by glossopharyngeal nerve and is tested using quinine on posterior third of the tongue.



Fig. 12.18: Hypoglossal nerve should be assessed in submandibular salivary gland enlargement.

Hypoglossal nerve function is checked by asking the patient to protrude the tongue out and observe the deviation of tongue. Accessory nerve function is assessed by asking the patient to shrug the shoulder, done in cases of enlarged upper deep cervical nodes infiltrating the nerve and paralysing the trapezius (**Fig. 12.18**).

Palpation of superficial temporal artery pulsation should be done in front of the tragus over the zygomatic bone.

Palpation of cervical nodes for significant enlargement should be done (**Figs 12.19A and B**). Features of Horner's syndrome should also be looked for in specific patients (**Fig. 12.20**).



Figs 12.19 A and B: Palpation of neck nodes in a patient with parotid swelling – submandibular and upper deep cervical.



Fig. 12.20: Cervical nodes should be palpated in submandibular salivary gland enlargement.

Features of facial nerve palsy

- Difficulty in chewing food as food accumulates in vestibule due to buccinator weakness (**Fig. 12.21**)
- Deviation of angle of mouth while talking, laughing, blowing, whistling due to paralysis of orbicularis oris (**Fig. 12.22**)
- Failure of closure of eyelids or easily opening of the eyelids after closure—paralysis of orbicularis oculi (**Figs 12.23A to C**)
- Absence of furrows while looking upwards—paralysis of frontal belly of occipitofrontalis
- Absence of corrugation in the forehead during frowning—paralysis of corrugator supercilii (**Fig. 12.24**)
- Deviation of angle of mouth towards opposite side—paralysis of levator anguli oris (**Fig. 12.25**)
- Loss of contraction of platysma in the neck while stretching the neck—paralysis of platysma (**Fig. 12.26**)
- Inability to blow the air by the check and on palpation reduced tone of buccinator—paralysis of buccinator
- Inability to whistle—paralysis of orbicularis oris

Proper diagnosis and investigations should be mentioned.

Investigations

X-ray of the part often intraoral X-ray to look for radiopaque stone in the submandibular region (**Fig. 12.27**).



Fig. 12.21: Facial nerve palsy – typical look.



Fig. 12.22: Inability to whistle – paralysis of orbicularis oris.

CT scan of the part including neck to see extent of the tumour, deep lobe involvement, and adjacent spread (**Fig. 12.28**).

FNAC of the swelling.

Sialography

Indications: Salivary fistulas; Sialectasis; Congenital conditions; Extraglandular masses. **Dye used is** Lipiodol or sodium diatrizoate (Hypaque). 24-gauge cannula is passed into either the Stensen's duct or Wharton's duct and 1 ml of the dye is injected and X-ray is taken.



A



B



C

Figs 12.23A to C: Failure of closure of eyelids or easily opening of the eyelids after closure – paralysis of orbicularis oculi.

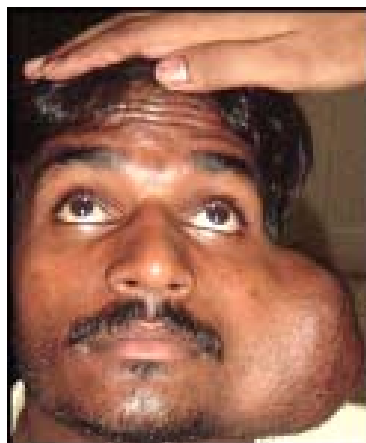


Fig. 12.24: Absence of corrugation in the forehead during frowning – paralysis of corrugator supercilii.

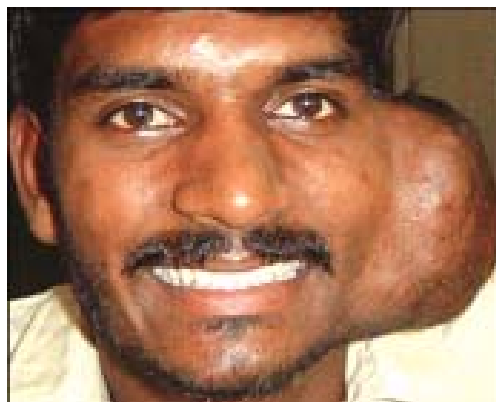


Fig. 12.25: Deviation of angle of mouth towards opposite side while clenching the teeth – paralysis of levator anguli oris.



Fig. 12.26: Loss of contraction of platysma in the neck while stretching the neck – paralysis of platysma.



Fig. 12.27: Plain X-ray showing submandibular salivary gland stone—radioopaque. (Courtesy by Dr Jagadishchandra MDS Mangalore).

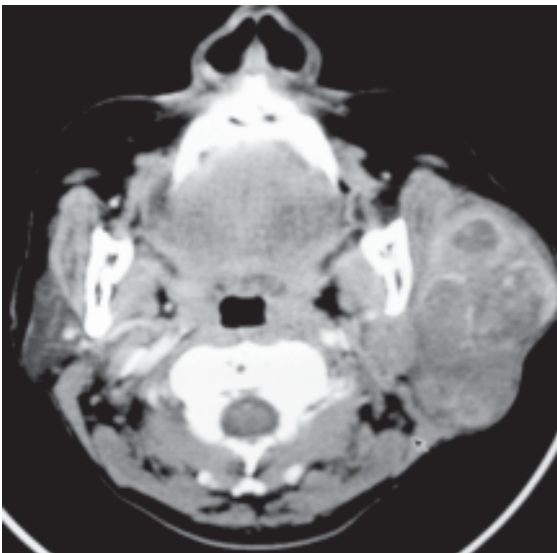


Fig. 12.28: CT scan of pleomorphic adenoma.

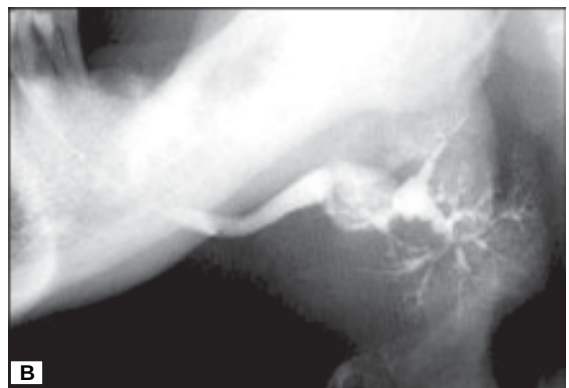
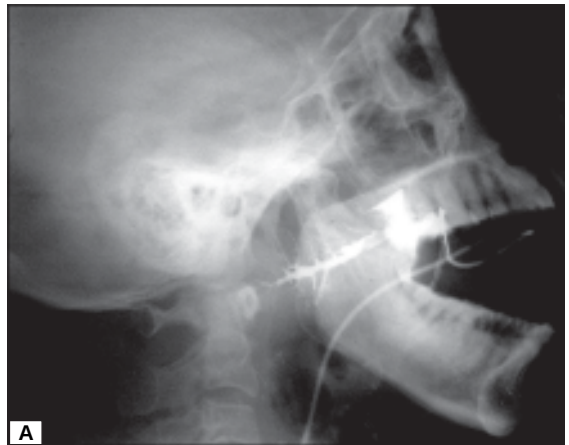
Findings: Narrowing (stricture); *grape-like* cluster appearance (sialectasis); dilatations; communications (Fistulas); mass lesions. *Sialography should never be performed in acute inflammation. Only one ml of dye is injected, if more dye is injected it causes extravasation and chemical sialadenitis (Figs 12.29A and B).*

Salivary Neoplasms

Classification

a. Epithelial:

1. Adenomas
 - Pleomorphic adenoma.



Figs 12.29A and B: Sialogram of parotid and submandibular salivary glands.

- Monomorphic adenomas.
 - Adenolymphoma (Warthin's tumour).
 - Oxyphil adenomas, oncocytoma.
 - Basal cell adenoma.

2. Carcinomas

- Mucoepidermoid carcinoma—Commonest malignancy.
- Acinic cell carcinoma 1%.
- Adenoid cystic carcinoma—Very aggressive.
- Adenocarcinoma.
- Squamous cell carcinoma - 2%.
- Carcinoma in ex. pleomorphic adenoma.
- Undifferentiated carcinoma.

b. Nonepithelial:

- Haemangioma—commonly seen in infants, usually in parotids. Spontaneous regression is common.
- Lymphangioma:
- Neurofibromas and neurilemmomas.

- c. **Malignant lymphomas**—Common in parotid; NHL type.
- d. **Secondary tumours** from head, neck region; bronchus and skin.
- e. **Lymphoepithelial tumours**—Benign type (5%) is common in females; can be bilateral (*Godwin's tumour*). Malignant is rare tumour—occurs in parotid and submandibular salivary glands (*Eskimoma*).

Incidence

75-80% salivary neoplasms are in the parotids of which 80% are benign; 80% of these are pleomorphic adenomas. 15% of salivary tumours are in the submandibular salivary gland; of which 60% are benign; 95% of these are pleomorphic adenomas. 10% of salivary neoplasms are in the minor salivary glands—palate, lips, cheeks and sublingual glands. Of these only 10% are benign (**Fig. 12.30**).



Fig. 12.30: Parotid gland enlargement in young boy.

Note: Parotid tumours are common but only 20% are malignant. Submandibular tumours are uncommon and 50% of them are malignant. Minor salivary gland tumours (other than sublingual glands) are rare and 90% of them are malignant. Sublingual salivary tumours are very rare but almost all sublingual salivary tumours are malignant.

Pleomorphic Adenomas (Mixed Salivary Tumour)

It is the commonest of the salivary gland tumour. It is 80% common. It is more common in parotids. Mixed

parotid tumour often begins in front of the tragus. It is mesenchymal, myoepithelial and duct reserve cell origin. **Grossly** it contains cartilages, cystic spaces, and solid tissues. **Histologically** it shows—Epithelial cells; myoepithelial cells; mucoid material with myxomatous changes; cartilages. Even though it is capsulated, tumour may come out as pseudopods and may extend beyond the main limit of the tumour tissue. When disease occurs in parotid, often it involves superficial lobe or superficial and deep lobe together (**Fig. 12.31**). But sometimes only deep lobe is involved where it presents as swelling in the lateral wall of the pharynx, soft palate and posterior pillar of the fauces. There may not be any visible swelling in the preauricular region—*Dumb bell tumour*. This tumour is in relation to styloid process, mandible, stylohyoid, styloglossus, stylopharyngeus muscles.



Fig. 12.31: Raised ear lobule is important sign of parotid enlargement.

Clinical Features

1:1 male to female ratio; 80% common; occurs in any age group; usually unilateral. Present as a single painless, smooth, firm lobulated, mobile swelling in front of the parotid with *positive curtain sign* (As the deep fascia is attached above to the zygomatic bone, it acts as a curtain, not allowing the parotid swelling to move above that level. Any swelling superficial to the deep fascia will move above the zygomatic bone). The ear lobule is lifted. When deep lobe is involved, swelling is commonly located in the lateral wall of pharynx, posterior pillar and over the soft palate. Facial nerve is not involved.

Long standing pleomorphic adenoma may turn into carcinoma – (carcinoma in ex. pleomorphic adenoma). Its features are: Recent increase in size; pain and nodularity; involvement of skin; involvement of masseter; involvement of facial nerve—lower facial nerve palsy; involvement of neck lymph node. Recurrence of pleomorphic adenoma is 5-40%; it is more if enucleated but less if parotidectomy is done. Malignant transformation is 3-5%; it may be 10% in long standing (15 years or more) pleomorphic adenomas.

Investigations

FNAC is very important and diagnostic. CT scan to know the status of deep lobe. Incision biopsy of parotid is contraindicated as there is chance of seedling of tumour and also injuring the facial nerve.

Adenolymphoma (Warthin's Tumour, Papillary cystadenolymphomatosum)

It is a benign tumour that occurs only in parotid, usually in the lower pole / near angle of the mandible; common in males; it is often bilateral – 10%; It is said to be due to trapping of jugular lymph sacs in parotid during developmental period. It is composed of double layered of columnar epithelium, with papillary projections into cystic spaces with lymphoid tissues in the stroma (Fig. 12.32).

Clinical Features

It presents as a slow growing, smooth, soft, cystic, fluctuant swelling, in the lower pole, often bilateral and nontender. It is common in males (4:1). It is not seen in Negroes. It is 10% common in old people – 60 years.

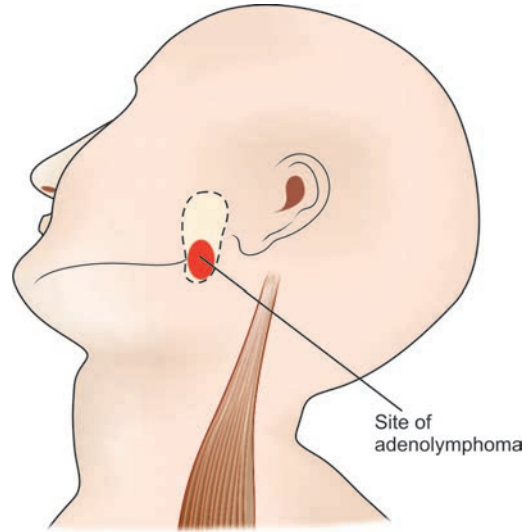


Fig. 12.32: Location of Warthin's adenolymphoma.

Investigations

Adenolymphoma produces a 'hot spot' in ^{99}Tc Technetium pertechnetate scan - it is diagnostic. FNAC. Adenolymphoma does not turn into malignancy.

Mucoepidermoid Tumour

It is the commonest malignant salivary gland tumour (in major salivary glands). It is slowly progressive, often attains a large size and spreads to neck lymph nodes. It contains malignant epidermoid and mucus secreting cells.

Types: Low grade and High grade. Facial nerve involvement is rare or very late in mucoepidermoid carcinoma of parotid.

TNM staging of malignant salivary tumours

T	— Tumour	N	— Lymph node
TX	— Tumour cannot be assessed	Nx	— Nodes not assessed
T0	— No evidence of primary tumour	N0	— Regional nodes not involved
T1	— Tumour < 2 cm without extraparenchymal spread	N1	— Single ipsilateral node < 3 cm
T2	— Tumour 2-4 cm	N2a	— Single ipsilateral node 3-6 cm
T3	— Tumour 4-6 cm	N2b	— Multiple ipsilateral nodes < 6 cm
	— or with extraparenchymal spread	N2c	— Bilateral or contralateral nodes < 6 cm
	— but no facial nerve spread	N3	— Single node spread > 6 cm.
T4	— Tumour > 6 cm		
	— or facial nerve spread	M	— Metastases
	— or base of skull spread.	M0	— No blood spread
		M1	— Blood spread present.

Clinical features: Swelling in the salivary (parotid or submandibular) region, slowly increasing in size, eventually attaining a large size, which is hard, nodular, often with involvement of skin and lymph nodes.

Adenoid Cystic Carcinoma (10% of Salivary Tumours)

It is common in minor salivary glands. It consists of myoepithelial cells and duct epithelial cells with cribriform or lace-like appearance. It involves facial nerve very early, spreads through the perineural sheath over a long distance more proximally and infiltrates into the perineural tissues and bone marrow. It also invades periosteum and bone medulla early and spreads extensively. It carries poor prognosis.

Acinic Cell Tumour

It is a rare, slow growing tumour that occurs almost always in parotid and is composed of cells alike serous acini. It is more common in women. It occurs in adult and elderly. It can involve facial nerve or neck lymph nodes. Clinically it is of variable consistency with soft and cystic areas. It is low grade malignant tumour.

GENERAL FEATURES OF MALIGNANT SALIVARY TUMOURS:

- Fixation, resorption of adjacent bone, pain and anaesthesia in the skin and mucosa
- Muscle paralysis, skin involvement and nodularity
- Involvement of jaw and masticatory muscle
- Nerve involvement (facial nerve in parotid or hypoglossal nerve in submandibular salivary gland)
- Mandibular branch of 5th cranial nerve may be involved when tumour tracks along the auriculotemporal nerve to the base of the skull causing severe pain in the distribution area
- Blood spread to lungs can occur

Clinical Syndromes Related to Involvement of Base of Skull in Malignant Parotid Tumour

Villaret-Mackenzie-Tapia syndrome of posterior retroparotid space: It is due to compression of the 9th to 12th cranial nerves and cervical sympathetic chain at the base of skull. There is dysphagia; dyspnoea; salivation changes; taste changes; weakness of trapezius, sternomastoid, same side tongue and soft palate; Horner's syndrome.

Vernet syndrome: It is due to compression at jugular foramen and results in 9th, 10th and 11th nerves.

Collet-Sicard syndrome: It is due to compression at posterior condylar space causing 9th, 10th, 11th and 12th nerve palsies.

Submandibular Salivary Gland Tumours

Benign Tumours

Benign tumours commonly pleomorphic adenomas are smooth, firm or hard, bidigitally palpable, without involving adjacent muscles or hypoglossal nerve or mandible bone. Diagnosis is by FNAC, Orthopantomogram and CT scan.

Malignant Tumours

Malignant tumours of submandibular salivary gland: They are hard, nodular, often get fixed to skin, muscles, hypoglossal nerve, and mandible. Diagnosis is by FNAC of primary tumour and of lymph nodes when involved, CT scan and OPG.

Specific Investigations

FNAC; CT scan to look for the involvement of deep lobe of the parotid; look for the involvement of bone, extension into the base of the skull, relation of tumour to internal carotid artery, styloid process, etc. (**Fig. 12.33**). OPG; Blood grouping and cross matching;



Fig. 12.33: Submandibular salivary gland tumour. Here oral cavity should be examined for deep lobe (bidigitally with one finger in the floor of the mouth); Wharton's duct (on either side of the frenulum of tongue); mandibular bone for thickening; hypoglossal nerve and lingual nerve palsy; neck nodes for spread.

required amount of blood is kept ready. FNAC of lymph node; MRI shows better soft tissue definition than CT scan. *Sialogram is not useful in assessment of tumour.*

Minor Salivary Gland Tumours

It is 10% of salivary tumours. It is common in—palate (40%); lip; cheek; sublingual glands. *Palate* is the commonest site (**Fig. 12.34**). 10% are benign—commonly *pleomorphic adenomas*. 90% are malignant—commonly *adenoid cystic carcinomas*. They present as swelling with ulcer over the summit. If it is malignant, then extension into the palate, maxilla, pterygoids can occur often with involvement of lymph node.



Fig. 12.34: Minor salivary gland tumour in the palate. Palate is the commonest site.

Differential diagnosis: Squamous cell carcinoma of oral cavity.

Investigations: Incision biopsy; CT Scan; X-ray maxilla; FNAC of lymph node.

Salivary gland tumours are usually benign in adult. It is rare in children but when it occurs, it is commonly malignant. Clinical and FNAC are diagnostic methods. *Open biopsy is contraindicated.* *Sialogram is not useful in salivary tumours.* CT or MRI are often needed. Nerve should be preserved in benign lesions. Nerve can be sacrificed to achieve clearance in malignancies.

Parotid Lymphoma

Parotid lymphoma can occur from the lymph nodes in the gland or from parotid parenchyma. It can occur in HIV patients; lymphoepithelial diseases and in Sjogren's syndrome. It is common in elderly. Disease may be confined to parotid gland or may involve other nodes in neck, mediastinum. 90% of salivary lymphomas occur in parotid. When it is confined to parotid total parotidectomy with radiotherapy and later chemotherapy is the treatment. When many other nodes are involved chemotherapy is the choice of therapy. *Note:* Lymphoma occasionally can occur in other salivary glands also (10% of all salivary lymphomas).

Complications of Surgery

Haemorrhage; infection; fistula; Frey's syndrome; facial nerve palsy; facial numbness; numbness in ear lobule due to injury to great auricular nerve; sialocele.

Frey's Syndrome (Auriculotemporal syndrome; gustatory sweating [Lucie Frey—Polish surgeon 1932])

It occurs in 10% of cases. It is due to injury to the auriculotemporal nerve, where in post ganglionic parasympathetic fibres from the otic ganglion become united to sympathetic nerves from the superior cervical ganglion (*Pseudosynapsis*). Auriculotemporal nerve has got two branches. Auricular branch supplies external acoustic meatus, surface of tympanic membrane, skin of auricle above external acoustic meatus. Temporal branch supplies hairy skin of the temple. Sweating and hyperesthesia occurs in this area of skin.

Causes: (1) Surgeries or accidental injuries to the parotid. (2) Surgeries or accidental injuries to temporomandibular joint.

Features: Flushing, sweating, pain and hyperaesthesia in the skin over the face innervated by the auriculotemporal nerve, whenever salivation is stimulated (i.e. during mastication). Condition causes real inconvenience to the patient. Starch iodine test will show the area blue (involved skin is painted with iodine and dried; dry starch applied over this area will turn blue due to more sweat in the area in Frey's syndrome).

Salivary Calculus and Sialadenitis

80% are in submandibular; 80% are radio-opaque; it is commonly calcium phosphate and calcium carbonate stones. Calculi in submandibular gland is more common, because the gland secretion is viscous, contains more calcium and also its drainage is nondependent, causing stasis. Secretion from parotid is serous, contains less calcium and so stones are not common.

Presentation

Acute features: Pain, swelling, tenderness is seen in submandibular region and floor of the mouth; Duct is inflamed and swollen. **Features in chronic cases:** Pain is more during mastication due to stimulation. Salivary secretion is more during mastication causing increase in gland size. Firm, tender swelling is palpable bidigitally. When stone is in the duct, it is palpable in the floor of the mouth as a tender swelling with features of inflammation in the duct. Pus exudes through the duct orifice. In submandibular salivary gland, the stones are multiple, with inflammation of gland (**sialadenitis**) (**Fig. 12.35**).

Differential Diagnosis

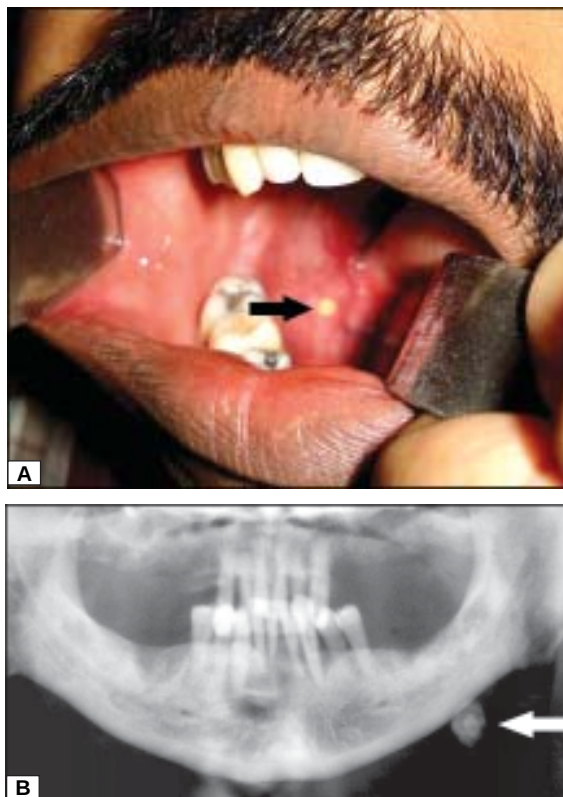
Submandibular lymphadenitis; salivary neoplasm.

Investigations

Intraoral X-ray (dental occlusion films) to see radio opaque stones (**Figs 12.36A and B**); FNAC of the gland to rule out other pathology; Total count and ESR in acute phase.



Fig. 12.35: Submandibular sialadenitis.



Figs 12.36A and B: Submandibular salivary stone in the duct and radioopaque shadow in X-ray.

Salivary calculi in submandibular gland	Salivary calculi in parotid gland
80% common	20% incidence (<i>Rare</i>)
80% Radio-opaque	Radiolucent
Seen in plain X-ray (intraoral)	Not seen in plain X-ray
Sialogram is not needed	Identified by sialogram

Calculi are common in submandibular salivary gland, because— • Viscous nature and mucin content • Calcium content • Non dependent drainage • Stasis.
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Sialosis

It is enlargement of the salivary gland due to fatty infiltration as a result of various metabolic causes like diabetes, acromegaly, obesity, liver disease. **Clinical features:** Bilateral diffuse enlargement of parotids, which is smooth, firm, nontender.

Causes for submandibular sialadenitis	Types of sialadenitis
Bacterial – more common. It is usually due to obstruction and stasis Trauma over duct causing oedema / stricture and stasis Viral – mumps – rare	Acute - Bacterial—occurs in submandibular salivary ductal obstruction (Wharton's) or in parotid gland. In parotid suppuration can occur leading into parotid abscess - Viral—common in parotid Chronic —common after partial obstruction of submandibular gland duct or due to stones in submandibular gland or hilum proximal to the level of crossing of the lingual nerve over the duct

Note: Salivary colic can be induced by meals, lemon juice, etc. Irritation of the lingual nerve, which is in very close proximity to submandibular salivary duct, causes referred pain in tongue – *lingual colic*.

Sialectasis

It is an aseptic dilatation of salivary ductules causing *grape-like* (cluster like) dilatations. It is a disease of unknown etiology with destruction of parenchyma of gland accompanied by stenosis and cyst formation in the ducts. It is common in parotids; often bilateral; presents as a smooth, soft, fluctuant, nontransilluminating swelling which increases in size during mastication. It is tender initially. It lasts for many days with a long symptom free period of the disease. Sialogram is diagnostic (*grape cluster* look).

Recurrent Childhood Parotitis

It is a recurrent, rapid enlargement of one or both parotids with fever and malaise in children of age group

between 3-6 years without any known etiology. Recurrent episodes with a quiescent period in between are typical. Sialogram shows *snowstorm punctate* sialectasis. Low dose antibiotics for long period may be required. Occasionally patient may need total conservative parotidectomy especially if it occurs late in adolescent period.

Parotid Abscess (Suppurative Sialadenitis)

It is a result of an acute *bacterial sialadenitis* of parotid gland. It is an ascending bacterial parotitis, due to reduced salivary flow and poor oral hygiene. Causative organisms are *Staphylococcus aureus*, *Streptococcus viridans*, and often others like gram-negative and anaerobic organisms. It is an ascending bacterial

Causes of acute parotitis (Differential diagnosis of suppurative parotitis)	Complications of parotid abscess
Viral—Mumps (commonest cause of parotitis), Coxsackie virus A and B, parainfluenza 1 and 3, Echo and lymphocytic choriomeningitis Bacterial – <i>Staphylococcus aureus</i> Allergic; HIV infection Radiotherapy, postoperative period Specific infections like syphilis Sjogren's syndrome often causing bilateral parotitis Features of acute parotitis Continuous, throbbing pain radiating to ear and side of the head; speaking/eating/any movements of TM joint is painful Fever with chills and rigors Diffuse swelling in front and behind the ear which is tender smooth firm with brawny induration, redness and warmth; it is nonmobile becomes prominent by clinching teeth Neck upper deep nodes may be tender and enlarged Restricted TM joint mobility; trismus Facial nerve is normal Oedematous ductal orifice with often discharge is common	Septicaemia Severe trismus Dysphagia Rupture into external auditory canal Chronic parotitis It can occur due to stone blocking the Stenson's duct presenting as rubbery hard slightly tender recurrent swelling in parotid region which is more during eating, with aching pain. Often it may be bilateral.

Remember about salivary fistula

Commonly from the parotid

It can be *internal* draining into the mouth or can be *external* draining outside

It is *acquired commonly* but rarely can be congenital

It can be due to surgery, trauma or due to sepsis

Fistula arising from the *gland parenchyma* drains through suture line but usually closes spontaneously. Leakage will be more during meals. Saliva is confirmed by its high amylase content compared to seroma/serous fluid

Fistula due to *ductal disruption* leaks profusely and invariably needs surgery to close it

Submandibular gland fistula commonly closes spontaneously, rarely if not, requires complete removal of the gland

Anticholinergics, irradiation, denervation of the gland, duct ligation are done to reduce saliva production

Excision of fistula, repair of the duct, diversion of the duct into the mouth are other options

In severe intractable cases, removal of the gland/total conservative parotidectomy is needed

parotitis, due to reduced salivary flow, dehydration, starvation, sepsis, after major surgery, radiotherapy for oral malignancies and poor oral hygiene. Parotid fascia is densely thick and tough and so parotid abscess does not show any fluctuation until very late stage.

Clinical features: Pyrexia, malaise, pain, trismus; firm swelling is seen in the parotid region which is red, tender, warm, well localised, tender lymph nodes are palpable in neck; Features of bacteraemia are present in severe cases. Pus or cloudy turbid saliva may be expressed from the parotid duct opening.

Investigations: U/S of parotid region; Pus collected from duct orifice is sent for culture and sensitivity; Needle aspiration from the abscess to confirm the formation of pus; *Sialogram is contraindicated in acute phase*, as it will cause retrograde infection leading into bacteraemia. *Note: In suppurative parotitis patient may develop severe laryngeal or pharyngeal oedema and may require steroids, tracheostomy and critical care.*

Parotid Fistula

Parotid fistula may arise from *parotid gland* or *duct* or *ductules*. It may open inside the mouth as internal fistula; or open outside onto the skin as external fistula (**Fig. 12.37**). Fistula from the duct has profuse discharge. Fistula from the gland often shows only minimal discharge.

Causes: After superficial parotidectomy; after drainage of parotid abscess; trauma; malignant recurrence of tumour.

Clinical features: Discharging fistula in the parotid region of face; tenderness and induration; trismus.



Fig. 12.37: Parotid fistula.

Diagnosis: Fistulogram. Sialography to find out the origin of the fistula whether from parotid gland or duct or ductules; discharge study; MRI.

Sjogren's Syndrome (Tage Sjogren, Swedish Physician 1939)

It is an autoimmune disease causing progressive destruction of salivary and lacrimal glands, leading to *keratoconjunctivitis sicca* (dry eyes) and *xerophthalmia* (dry mouth). **Types: 1. Primary, 2. Secondary.** *Secondary Sjogren's syndrome:* Dry mouth; Dry eyes; With association of connective tissue disorders like primary biliary cirrhosis (near 100%); SLE (30%); Rheumatoid arthritis (15%). Female to male ratio is 10:1.

Primary Sjogren's syndrome: Severe dry mouth; Severe dry eyes; Widespread dysfunction of exocrine glands; incidence of developing lymphomas is high; there is no association of connective tissue disorders.

Clinical Features

It is common in middle aged females who present with dry eyes, dry mouth, enlarged parotids and enlarged lacrimal glands; often they are tender; superadded infection of the mouth with, *Candida albicans* is common. Sjogren's syndrome often causes bilateral parotitis.

Investigations

Autoantibody estimation–Rheumatoid factor, anti-nuclear factor, salivary duct antibody; Sialography; estimation of salivary flow; slit-lamp test of eyes; Schirmer test – to detect lack of lacrimal secretion; FNAC of parotids and lacrimal glands; ⁹⁹Tc pertechnetate scan for gland function.

Mikulicz Disease

It is a clinical variant of Sjogren's syndrome. It is an autoimmune disorder of salivary and lacrimal glands, resulting in infiltration of the glands with round cells. Glandular tissue is replaced by lymphocytes.

Triad: (1) Symmetrical and progressive enlargement of all salivary glands (parotid, submandibular, sublingual, accessory parotid). (2) Narrowing of palpebral fissures due to enlargement of the lacrimal glands. (3) Parchment-like dryness of the mouth but patient is *not thirsty*.

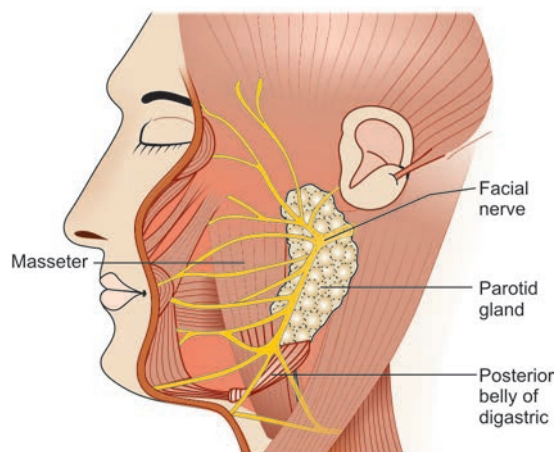
Heerfordt's Syndrome

It is sarcoidosis of parotid swelling; anterior uveitis; facial palsy and fever.

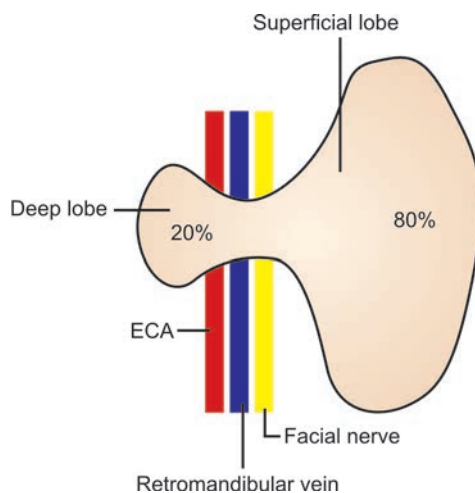
Anatomy of Parotid Gland (Para-around, Otis-ear)

Parts of the Parotid Gland

Superficial part (80%) - lies over the posterior part of the ramus of mandible. Deep part lies behind the mandible and medial pterygoid muscle. Parotid gland is pyramidal shaped with upper pole just below the zygomatic bone and wedged between external auditory meatus and the mandibular joint. Anterior border is over the masseter; lower pole is below and behind the angle of the mandible and indented by sternomastoid. Parotid is covered by dense parotid fascia which is derived from investing layer of deep fascia (Figs 12.38A and B). **Accessory parotid** is prolon-



[A]



[B]

Figs 12.38A and B: Anatomical relations of the parotid gland.

gation of the gland along the parotid duct. Parotid (*Stensen's*) duct is 2-3 mm in diameter, emerges from the anterior border of the gland runs horizontally across masseter and passes through the buccinator muscle and opens into the oral mucosa opposite upper second molar tooth (Fig. 12.39). **Facial nerve** emerges from the stylomastoid foramen lying between external auditory meatus and mastoid process. It passes around the neck of the condyle of mandible and becomes superficial, later dividing into *temporofacial* and *cervicofacial* branches which in turn divides into many branches. Some of these may be interconnected as *pes anserinus* (goose foot) (Fig. 12.40). Branches are



Fig. 12.39: Accessory parotid tumour. It contains both serous and mucous acini.

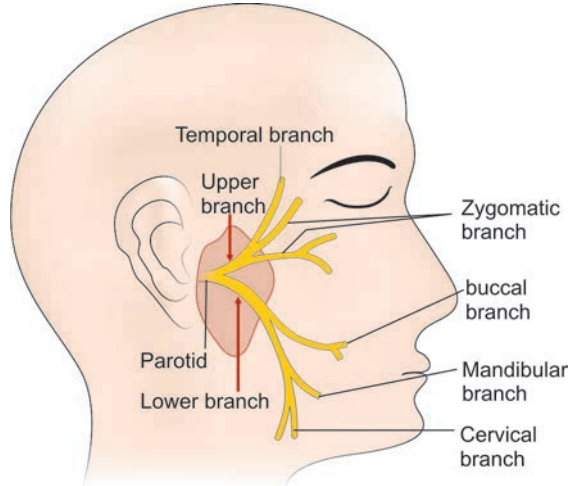


Fig. 12.40: Facial nerve distribution – Pes anserinus.

– temporal (auricularis anterior and superior part of frontalis), zygomatic (frontalis and orbicularis oculi), upper buccal and lower buccal (buccinator, orbicularis oris, elevators of the lip), mandibular (lower lip muscles) and cervical (platysma) (**Fig. 12.41**).

Blood supply is from external carotid artery; venous drainage is to external jugular vein. Nerve supply is

from autonomic nervous system; parasympathetic is secretomotor from auriculotemporal nerve; sympathetic is vasomotor from plexus around external carotid artery. Faciovenous plane of Patey of retromandibular vein is of surgical importance as facial nerve branches lie superficial to it. 25% of saliva is from parotids.

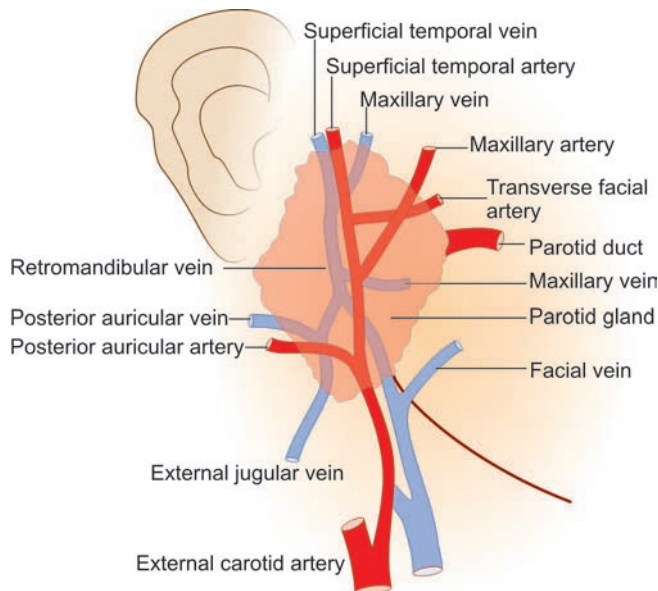


Fig. 12.41: Patey's vascular plane in parotid.

Structures within the parotid gland from deep to superficial–

External carotid artery, maxillary artery, superficial temporal artery, posterior auricular artery
 Retromandibular vein (by maxillary and superficial temporal veins)
 Facial nerve with its branches.

Great auricular nerve (cutaneous sensory around angle and lower part of the ear lobule) and auriculo-temporal nerve which is from mandibular division of trigeminal nerve (secretomotor to parotid gland) are other nerves present in relation to parotid gland.

Secretomotor Fibres

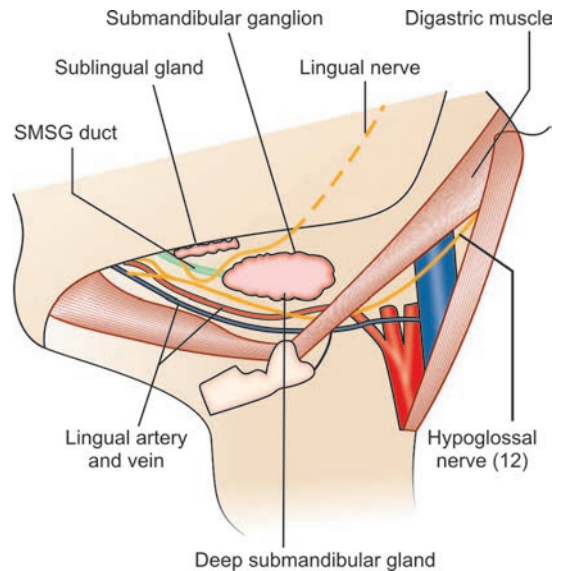
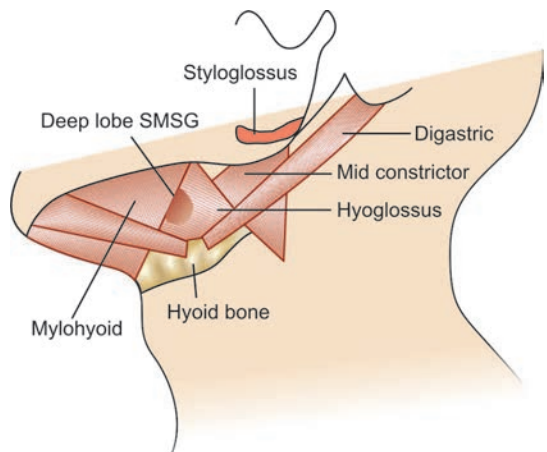
Secretomotor preganglionic fibres from *inferior salivary nucleus* → glossopharyngeal nerve → tympanic branch → tympanic plexus → lesser superficial petrosal nerve → otic ganglion → post-ganglionic fibres → auriculotemporal nerve, branch of mandibular division of trigeminal nerve → parotid gland.

Parotid gland is serous. Submandibular gland is mixed (major is mucous). Sublingual is mucous. Minor salivary glands are mucous except *von Eber's glands* which empty into the circumvallate papillae and glands in the tongue tip.

SUBMANDIBULAR SALIVARY GLAND**Parts**

Superficial part lies in submandibular triangle, superficial to mylohyoid and hyoglossus muscles, between the two bellies of digastric muscles. *Deep part* is in the floor of the mouth and deep to the mylohyoid. Submandibular (*Wharton's*) duct (5 cm), comes from the deep part of the gland, enters the floor of the mouth, on a papilla beside the frenum of the tongue. Lingual nerve and submandibular ganglion are attached to upper pole of the gland. Facial artery emerges from under surface of the stylohyoid muscle, enters the gland from posterior and deep surface reaching its lateral surface crossing the lower border of mandible to enter the face. Venous drainage is to anterior facial vein. 70% of total saliva is from submandibular salivary gland (**Figs 12.42A and B**).

Resting salivary flow usually arises from the submandibular salivary gland. *Sialorrhoea* is increased

**A****B**

Figs 12.42A and B: Anatomical relations of the submandibular salivary gland.

salivary flow often seen due to drugs, in cerebral palsy, physically handicapped person, children, and psychiatry patients. Intractable sialorrhoea can be corrected by different surgeries to submandibular salivary gland like duct repositioning to excision of the gland. Normal salivary secretion per day is 1500 ml. It is hypotonic fluid with pH 7.0. It contains α amylase.

Xerostomia is decreased salivary flow. It is seen in post-menopausal women, depression, dehydration, use of antidepressant drugs; anticholinergic drugs, Sjogren's syndrome, radiotherapy to head and neck region.

Secretomotor Fibres of Submandibular Salivary Gland

Preganglionic fibres from *superior salivary nucleus* → facial nerve → chorda tympani nerve → lingual nerve → submandibular ganglion → post-ganglionic fibres → submandibular and sublingual salivary glands.

Minor Salivary Glands

There are around 450 minor salivary glands which are distributed in lips, cheeks, palate and floor of the mouth. Glands also may be present in oropharynx, larynx, trachea and paranasal sinuses. They contribute to 10% of total salivary volume. Sublingual salivary glands are minor salivary glands one on each side; located in the anterior aspect of the floor of the mouth in relation to mucosa, mylohyoid muscle, body of the mandible near mental symphysis (**Fig. 12.43**). Gland drains directly into mucosa or through a duct which drains into submandibular duct. This duct is called as *Bartholin duct*. Mikulicz's disease is common in sublingual salivary gland. Minor salivary glands are *not present* in gingivae and anterior portion of the hard palate.



Fig. 12.43: Swelling in sublingual region.

Ectopic Salivary Gland

Ectopic salivary gland also called as aberrant salivary gland / migrant salivary gland is nothing but ectopic lobe of the juxtaposed salivary gland. It is commonly seen in relation to submandibular salivary gland. Commonest ectopic salivary tissue is *Stafne bone cyst*. It is invagination of the juxtaposed submandibular salivary gland into the mandible bone on its lingual aspect. X-ray shows radiolucent area due to the cyst below the angle of the mandible, lower to inferior dental vessels and nerve. Jaws, eyelids, middle ear, paranasal sinus, nose, rarely skin of face and neck are other sites wherein ectopic salivary tissue can be demonstrated.

Examination of Neck

Neck is a complex anatomical area comprised of many compartments, triangles, tubes (trachea, oesophagus), vessels and lymph nodes. Thorough anatomical knowledge of the area is essential for safe clinical and surgical practice. Student should read the specific anatomical book for the same.

History taking begins with:

Name:

Age:

Occupation:

Address:

Sex:

Cystic hygroma, branchial cyst and fistula are congenital in origin. Sternomastoid tumour, a misnomer seen in infants and children, due to organised haematoma in sternomastoid muscle leading to fibrosis of its muscle fibres following a birth trauma. Tuberculous lymphadenitis occurs in young adults; carcinoma secondaries in lymph nodes usually occur in elderly.

History

History of Present Illness

Swelling: Swelling is the commonest presentation in the neck. Lymph nodal mass is the commonest type of swelling in the neck. It could be due to lymphadenitis (nonspecific bacterial infection and inflammation); tuberculosis; malignancy; AIDS, viral causes. Other swellings which can occur in the neck are cystic swellings, carotid body tumour, cervical rib, carotid aneurysm, etc. History (like in chapter swelling) should be asked in detail. History associated with onset, progress, duration, recent increase in size, number, etc. should be asked. Acute inflammatory swellings are of very short duration with signs of acute inflammation. Swelling of short duration is commonly

malignant. Malignancy may be lymph node secondaries or lymphoma. It takes few months for tuberculous cold abscess to evolve in a tuberculous lymphadenitis. Presence of similar swelling elsewhere in the body like in axilla, abdomen, and groin suggests that it could be lymphoma.

Pain: Time of onset of pain, whether it was present at the beginning, whether initially painless later became painful (sepsis, infiltration, tumour necrosis). Acute conditions are painful to start. Malignancy is initially painless.

Fever: Fever suggests acute inflammatory condition; mild fever with occasionally evening rise is seen in tuberculous lymphadenitis. But one should remember that fever is not necessarily a feature in all patients with many tuberculous lymphadenitis.

Relevant histories like cough, haemoptysis (tuberculosis, lymphoma, and carcinoma), voice change, dyspnoea, dysphagia, abdominal discomfort are important to be noted.

Past History

Past history of treatment for tuberculosis, their details, treatment for malignancy (surgery, chemotherapy, radiotherapy) are important points should be asked.

Personal History

History of smoking, alcohol consumption, dietary habits, decreased appetite and loss of weight (in advanced carcinoma lymphoma and in tuberculosis) should be asked.

General Examination

Anaemia, clubbing, jaundice are checked. Pulse and blood pressure are recorded, nutrition and built are assessed.

Local Examination

Inspection

Neck should be examined with proper exposure upto the nipples. Entire neck including all triangles should be examined (**Fig. 13.1**).



Fig. 13.1: Proper inspection of the neck is essential.

Swelling: Swelling is the commonest presentation in the neck. Lymph nodes are the commonest of the neck swellings. Its number, site, size, shape, extent, surface, dilated veins, skin changes like redness, oedema, ulceration or fungation should be inspected. *Branchial cyst* is located at the level of upper 1/3rd and middle 1/3rd of the sternomastoid muscle with posterior 1/2 of the swelling lying under the sternomastoid muscle. Lymph nodes can get enlarged in any area in neck. Surface is nodular in secondaries and tuberculosis, smooth in lymphoma. Cold abscess shows smooth surface on inspection. *Ranula* can occur in upper neck. *Dermoid cyst* can occur in chin, in space of Burns in midline. *Cervical rib*, *cystic hygroma*, *subclavian artery aneurysm* occur in posterior triangle of the neck. *Carotid artery aneurysm* is seen usually in carotid triangle or along the line of carotid artery. *Carotid body tumour* is seen in carotid triangle. Swelling should be differentiated from thyroid swelling by checking *movement with deglutition*. Thyroid swelling, thyroglossal cyst, subhyoid bursa all move with deglutition.

Skin over the swelling is looked for—dilated veins, redness (inflammation), oedema (inflammation or

malignancy), discharging sinus/fistula, ulcer, scar. *Branchial fistula* is located in junction of middle 1/3rd and lower 1/3rd of the sternomastoid muscle along the anterior margin; *thyroglossal fistula* in the midline lower 1/3rd; tuberculous sinus can occur in the neck, the site depends on the location of the underlying tuberculous lymphadenitis, undermined tuberculous ulcer is known to occur; chronic pyogenic osteomyelitis of the mandible can cause discharging sinus over the lower margin of the mandible; actinomycosis of mandible causes multiple sinuses with discharge containing sulphur granule; syphilitic gummatous ulcer may be seen in sternomastoid muscle (now rare). Sinus, ulceration, fungation may be features of advanced fixed secondaries in the lymph node.

Wasting of trapezius, sternomastoid, and other neck muscles should be noted, *torticollis* (chin turns towards opposite side and neck towards same side due to spasm/contraction/fibrosis of sternomastoid muscle); dilated veins in neck, and chest wall suggest mediastinal compression by tumour/nodes. In *torticollis*, face is often less developed on the affected side. When patient attempts to straighten the neck, *sternal head* of sternomastoid stands out taut and firm with inability to straighten the head. Asymmetry of skull can be detected by examining the head and neck from behind. All swellings should be inspected carefully with relation to *sternomastoid muscle*.

Palpation

Palpation of neck is done with patient sitting on a stool and examiner standing behind the patient. First *always ascertain the relation of the swelling to sternomastoid* by palpation. With examiner standing behind the patient, patient is asked to push his chin against examiner's hand firmly to make the sternomastoid muscle tense; with the other hand examiner should palpate the sternomastoid muscle from below upward along its anterior border and ascertain the swelling in relation to the muscle (**Fig. 13.2**).

Cervical lymph nodes are also examined *from behind*. Patient should *flex* the neck to relax the muscle and fascia to make the swelling better for palpation. Usual order of lymph nodes are Levels I, II, III, IV, V and VI. Submandibular group of nodes are felt with neck flexed towards same side. In posterior triangle both supraclavicular and suboccipital nodes should



Fig. 13.2: Method of checking the relation of sternomastoid muscle to swelling by palpating from behind.

be palpated. Often supraclavicular lymph nodes are palpated from front. *Virchow's node* between the two heads of the sternomastoid is palpated from front. Swelling should be palpated for location, size, shape, surface, consistency, margin, reducibility, impulse on coughing, and mobility. Tuberculosis causes *matted* lymph nodes; secondaries cause *stony hard nodular* swelling; lymphoma causes *India rubber-like firm* swelling (**Figs 13.3 and 13.4A and B**).



Fig. 13.3: Hodgkin's lymphoma neck both sides. It is India rubber-like firm in consistency.



Figs 13.4A and B: Secondaries in neck nodes – nodular surface, stony hard consistency are typical.

Plane of the swelling is checked by contracting the sternomastoid against resistance by turning the chin opposite side (against the resistance of the examiner's hand). Examiner's hand is placed under the chin of the patient; who is asked to push/nod the chin downwards against resistance of examiner's hand and swelling is palpated to check whether it is deep to sternomastoid or not and the plane is checked on both sides simultaneously. If swelling is in deeper plane, it reduces in size with restricted mobility when muscle is made taut. If swelling is in superficial plane, it



Figs 13.5A to D: Skin pinching (for fixity); mobility; contraction of sternomastoid against resistance towards opposite side to find out the plane of the swelling; palpation from all directions of the swelling are essential.

becomes more prominent after muscle contraction and still mobile over the muscle (**Figs 13.5A to D**).

Swelling will be completely immobile if it is fixed posteriorly to paravertebral region as seen in advanced secondaries in neck.

Pinching the skin/gliding the skin over the swelling should be done to assess the fixity to skin. It can be

often fixed to swelling in secondaries/tuberculosis forming collar stud abscess and acute lymphadenitis (**Figs 13.6A to C**).

Pulsation: Expansile/transmitted, *fluctuation* (in two directions)/Paget's test; *transillumination* (like in other swelling) should be checked. Cold abscess, cystic hygroma, lymph cyst, branchial cyst, dermoid cyst,



Figs 13.6A to C: Cold abscess, eliciting fluctuation (Paget's) and collar stud abscess – due to tuberculosis.

subhyoid bursa, thyroglossal cyst are *fluctuant*. Cystic hygroma and lymph cyst are *brilliantly transilluminant*. *Laryngocele* may show expansile impulse or becomes prominent while blowing. Carotid aneurysm shows *expansile* pulsation; carotid body tumour shows *transmitted* pulsation. Nodular surface, hard consistency is seen in secondaries in lymph node. Smooth surface with firm, *India rubber consistency* is seen in lymphoma. Carotid body tumour and carotid aneurysm move *only horizontally* not in the line of the artery. Rare tumour (neurofibroma) can occur from vagus nerve on the posterior aspect of the carotid sheath which causes cough sensation while palpation; swelling is only horizontally mobile, firm, with typical transmitted pulsation.

Torticollis due to shortening of sternomastoid, should be differentiated from ocular torticollis. Head is clasped by examiner's hand and slowly straightened observing the eyes. Straightening of the head makes squint apparent in ocular torticollis.

Other relevant examinations to be done are—protrusion of tongue to look for hypoglossal nerve palsy (tongue deviates towards affected side); spinal accessory nerve (shrugging of shoulder is defective with wasting of trapezius) (**Fig. 13.7**); features of cervical sympathetic chain involvement (Horner's syndrome); carotid artery pulsation; superficial temporal artery pulsation; tracheal palpation; laryngeal crepitus (normally it is present, but absent in advanced laryngeal carcinoma) (**Figs 13.8A to 13.10**). *Cervical spine*



Fig. 13.7: Shrugging of shoulder against resistance to check trapezius paralysis due to infiltration of spinal accessory nerve.



Figs 13.8A and B: Laryngeal crepitus is present normally. It will be absent in advanced carcinoma larynx. Trachea also should be examined for deviation.



Fig. 13.9: Superficial temporal artery pulsation should be checked in front of tragus.



Fig. 13.10: Carotid pulsation should be checked to confirm whether it is infiltrated/encased by tumour or any presence of thrill (suggests stenosis) and also should be auscultated for bruit.

should be examined (paraspinal spasm, tenderness, deformity, movements) especially in case of tuberculosis for primary. In females (rarely in male) carcinoma breast may be the cause of neck node enlargement, so breast should be examined in suspected cases.

Oral cavity should be examined in all neck swellings especially when swelling is thought to be from lymph node. Tonsils may show tubercles in case of tubercular lymphadenitis. Retropharyngeal abscess in tuberculosis is chronic and lies in midline (**Fig. 13.11**).



Fig. 13.11: Oral cavity should be examined in all neck swelling patients thoroughly.

Other lymph nodes in the body should be examined—axillary, para-aortic, iliac, inguinal, epitrochlear (above the medial epicondyle and on medial aspect of the arm), and popliteal lymph nodes. Lymphoma may cause generalised lymphadenopathy.

Drainage area of the specific lymph nodes which are palpable should be examined. *Cervical lymph nodes* drain from lymphatics of head, neck, face, oral cavity, nasal cavity, paranasal sinuses, pharynx, larynx and thyroid. Left supraclavicular nodes receive from left upper limb, left side chest wall, left breast, abdomen and both testes. Cervical lymph nodes can be superficial or deep. Nodes are arranged in different levels (*Sloan Catering memorial hospital USA*) – *Level I to level VI. Level VII is mediastinal node.* Level I is submental and submandibular nodes; level II is upper deep cervical nodes; level III is middle deep cervical nodes; level IV is lower deep cervical nodes; level V is

posterior triangle nodes; level VI is central nodes (paratracheal and laryngeal). Level VII is mediastinal node. Level I and level II are further divided into a and b. Ia is submental nodes; Ib is submandibular nodes. Level IIa nodes lies below the spinal accessory nerve (in sternomastoid muscle) and IIb above the nerve; Level Va nodes are above the spinal accessory level (in posterior triangle) and level Vb is below it (**Figs 13.12 to 13.18**).

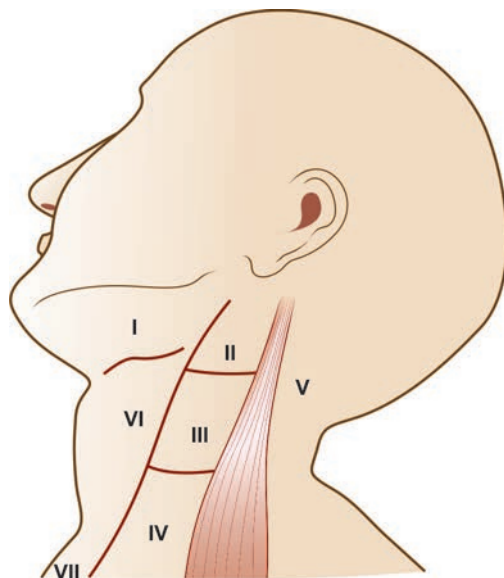


Fig. 13.12: Levels in cervical lymph nodes.

Inner Waldeyer's ring which includes adenoids, tubal tonsils, faucial tonsils, lingual tonsils also should be examined.

Outer Waldeyer's ring includes retropharyngeal lymph nodes; jugulodigastric lymph nodes; submandibular lymph nodes; submental nodes.

Healy's classification of lymph nodes in neck:

SH—superior horizontal chain; IH—inferior horizontal chain; PV—posterior vertical chain; IV—intermediate chain; AV—anterior vertical chain.

Percussion

In laryngocoele tympanic note may be heard on percussion. Percussion over the sternum is important to elicit tenderness in lymphoma (bone marrow involvement – Stage IV) and also in mediastinal nodal mass that will elicit dullness if present (**Fig. 13.19**).



Figs 13.13A and B: Submandibular lymph nodes are examined from behind with flexion of the neck. Bidigital palpation is done to differentiate lymph node from submandibular salivary gland. Lymph nodes are not bidigitally palpable; submandibular salivary gland is bidigitally palpable.



Fig. 13.14: Palpation of level 2 lymph nodes.



Fig. 13.15: Palpation of level 3 lymph nodes.



Fig. 13.16: Palpation of level 4 lymph nodes.



Fig. 13.18: Palpation of Virchow's lymph node in the neck in between two heads of sternomastoid.



A



B

Figs 13.17A and B: Palpation of level 5 lymph nodes. It is palpated both from behind and front.



Fig. 13.19: Percussion over sternum for tenderness (in lymphoma and lymphatic leukaemia) and dullness for mediastinal mass.

Auscultation

Auscultation is done to hear bruit over carotids in carotid artery aneurysm; over supraclavicular region in subclavian artery aneurysm.

Systemic Examination

Abdomen should be examined for splenomegaly and hepatomegaly in case of lymphoma; hepatomegaly in case of secondaries.

Respiratory system is examined for pulmonary tuberculosis, bronchogenic carcinoma.

Skeletal system – spine and long bones should be examined for secondaries and in case of lymphoma. Tenderness, swelling, pathological fracture may be evident. Neurological deficits and paraplegia with bowel and urinary incontinence may be present in case of spine involvement.

Examination of Nasopharynx, Oropharynx and Hypopharynx

Pharynx has got 3 parts—*Nasopharynx*; *oropharynx*; *laryngopharynx*. *Nasopharynx* is uppermost part of the pharynx situated behind the nose and above the lower border of the soft palate. Anteriorly it communicates with nasal cavities; inferiorly with *oropharynx* through nasopharyngeal isthmus (*Passavant's ridge*). Lateral wall contains opening of the auditory tube; tubal elevation; fossa of Rosenmuller/pharyngeal recess behind the tubal elevation. This is above the upper edge of superior constrictor. Roof continues as posterior wall of nasopharynx. Adjacent to base of occiput nasopharynx contains lymphoid aggregates called as *pharyngeal tonsil* which is small or absent in adult but well developed in children and *pathologically* can be enlarged as adenoids. *Tubal tonsil* is collection of lymphoid tissue one on each side behind the tubal opening. *Oropharynx* is middle part of the pharynx which communicates above to nasopharynx through nasopharyngeal isthmus, in front with the oral cavity through oropharyngeal isthmus (isthmus of fauces), below to laryngopharynx at the level of upper border of epiglottis. *Palatine tonsil* lies in tonsillar fossa in the lateral wall one on each side between palatopharyngeal arch (by palatopharyngeus muscle) behind, palatoglossus arch (palatoglossus muscle) in front. Tonsils are *seen per orally*. Oropharynx is formed behind by superior, middle and posterior constrictors of the pharynx. *Laryngopharynx* or *hypopharynx* is laryngeal part of the pharynx extends from the upper part of epiglottis above to lower margin of cricoid below. Anterior wall of hypopharynx shows laryngeal inlet, posterior surfaces of cricoid and arytenoids. Posterior wall is formed by constrictors. Middle constrictor overlaps the upper margin of inferior constrictor; superior constrictor overlaps middle constrictor in front (superficially). *Pyriform fossa* is

located in the lateral wall of the pharynx as a depression on each side of the laryngeal inlet; bonded medially by aryepiglottic fold, laterally by thyroid cartilage and thyrohyoid membrane.

Carcinoma pyriform fossa may be silent; or presents as difficulty in swallowing saliva as opposed to food, later definitive dysphagia, change in voice, laryngeal fixation (as late feature) or palpable significant cervical lymph nodes. It is beyond reach for digital examination. Laryngeal mirror is essential to visualise and examine it. *Sideropaenic dysphagia* and *postcricoid carcinoma* can also occur.

Oropharynx is examined using two spatulas. Tongue is depressed with one spatula and with another cheek is retracted laterally with its tip gently compressing the anterior pillar of the fauces. Tonsillar crypts, size, surface, discharge, surrounding areas should be examined. Often tonsils are enlarged so much that both sides touch in the midline. Tubercles in the tonsils may be obvious. Ear pain, halitosis, blood stained saliva, haemorrhage, ulceration, fungation, dysphagia, trismus, palpable significant neck lymph nodes are features of carcinoma of tonsils. Lymphosarcoma may develop in the tonsil in young individual. Painless swelling in throat, thick speech, large pale tonsil, are the initial features of lymphosarcoma of tonsil. Extracapsular spread may cause a palpable and often visible swelling behind and below the angle of the mandible as a direct extension of the primary tumour. But sooner cervical lymph nodes get involved in same place as secondaries and become palpable.

Nasopharynx is *palpated* with patient sitting in a stool. Examiner stands behind the patient with patient extending his neck and head is supported by examiner's body. One side index finger pushes the cheek inward from outside after opening the mouth (to prevent biting of the examiner's hand). Index finger of the other hand is passed inside, towards nasopharynx to sweep over the roof and walls of the nasopharynx (**Fig. 13.20**). Retropharyngeal abscess is always felt and only often seen after proper depression of the tongue (can be seen when inspected using a direct laryngoscope). It is felt as an indentable cushion-like projection to the finger. Acute retropharyngeal abscess is usually due to suppuration of retropharyngeal lymph node and occupies a lateral position. Chronic retropharyngeal abscess is usually due to tuberculosis of cervical spine



Fig. 13.20: Palpation of nasopharynx from behind.

(C₆) and is behind the prevertebral fascia and so situated in midline. However occasionally tuberculosis of retropharyngeal lymph nodes can occur as a rare entity and in such situation, it will be in lateral position. It also may present as swelling/cold abscess in the neck behind the sternomastoid muscle.

Examination of Nasal Cavities and Paranasal Air Sinuses

Nasal cavities should be examined using a nasal speculum. Frontal, ethmoidal and maxillary air sinuses should be examined for fullness, swelling, tenderness. Sinusitis is common. Tumours of maxillary and ethmoidal air sinuses should be thought of. Maxillary tumour causes upward displacement of eye; ethmoidal tumour causes lateral displacement of the eye. Neoplasm in frontal air sinuses is practically rare. Proper knowledge of surgical anatomy of these areas is essential.

Investigations

FNAC of the node. It is useful in secondaries, tuberculosis (epithelioid cells). In branchial cyst cholesterol crystals are seen (**Fig. 13.21**).

- Lymph node biopsy in suspected case of lymphoma.
- Chest X-ray, X-ray cervical spine in tuberculosis.
- Fistulogram in branchial fistula and other fistulas.
- MR fistulogram.
- CT scan Chest and neck for multiple nodal mass.



Fig. 13.21: FNAC of neck lymph node is ideal initial investigation.

- Barium swallow or water soluble contrast study (better) in pharyngeal pouch.
- Arterial Doppler in aneurysm, carotid body tumour, subclavian artery aneurysm.
- Carotid or subclavian arteriogram
- Discharge study for AFB, culture, cytology.
- Edge biopsy if ulcer is present.
- Laryngoscopy/bronchoscopy/mediastinoscopy/oesophagoscopy in relevant causes.

Branchial Cyst

It arises from the remnants of second branchial cleft. Normally 2nd, 3rd, 4th clefts disappear to form a smooth neck. Persistent 2nd cleft is called as *cervical sinus* which eventually gets sequestered to form *branchial cyst* (**Figs 13.22A and B**).

Features: It is a congenital swelling in the neck but presents in 2nd or 3rd decade. Swelling is located in the neck, beneath the anterior border of upper third of the sternomastoid muscle. It is smooth, soft/tensely cystic (and so hard), fluctuant, occasionally transilluminant. It feels like '*half filled double hot water bottle*'. Cyst is not compressible not reducible. Neck nodes are not enlarged. It contains fat globules and cholesterol

Different classifications of neck swellings

Midline swellings of the neck	Lateral swellings	Others
Ludwig's angina Submental lymph node Sublingual dermoid Thyroglossal cyst Subhyoid bursa Thyroid isthmus swelling Prelaryngeal and pretracheal lymph nodes Midline dermoids and lipomas Suprasternal lymph node	Submandibular triangle Submandibular salivary gland enlargement Submandibular lymph node enlargement Plunging ranula Jaw tumours extending down Carotid triangle Carotid aneurysm Carotid body tumour Branchial cyst Branchiogenic carcinoma Thyroid swelling – lateral lobe Sternomastoid tumour Lymph nodal mass Posterior triangle Lymph nodal mass Cystic hygroma Pharyngeal pouch Subclavian aneurysm Cervical rib Lateral aberrant thyroid	Acute Cellulitis Lymphadenitis Ludwig's angina Chronic Cystic Cold abscess Cystic lesions of thyroid Branchial cyst Thyroglossal cyst Cystic hygroma Dermoid cyst Sebaceous cyst Solid Secondaries in neck lymph nodes Thyroid swelling Branchiogenic carcinoma Sternomastoid tumour Cervical rib Soft tissue tumour Pulsatile Carotid aneurysm Carotid body tumour Subclavian artery aneurysm Primary toxic goitre



Figs 13.22A and B: Branchial cyst.

crystals. It is golden yellow in colour. Histologically, it is lined by *squamous epithelium*.

Differential diagnosis: Cold abscess, lymph cyst. It may get infected to form an abscess. FNAC shows *cholesterol crystals*. Cyst lies in relation with carotids, hypoglossal nerve, glossopharyngeal nerve, and spinal accessory nerve, posterior belly of digastric and pharyngeal wall. Medially it is close to the posterior pillar of tonsils. During dissection, all these structures should be taken care of.

Branchial Fistula

It is a persistent second branchial cleft with a communication outside to the exterior. It is commonly a congenital fistula. Occasionally the condition is secondary to incised, infected branchial cyst. Often it is bilateral (**Figs 13.23A and B**). *External orifice* of the fistula is situated in the *lower third of the neck* near the anterior border of the sternomastoid muscle (incised infected branchial cyst causes sinus in upper 1/3rd of neck). *Internal orifice* is located on the anterior aspect of the posterior pillar of the fauces, just behind the tonsils. Sometimes fistula ends internally as blind



Figs 13.23A and B: Branchial fistula.

end. Track is lined by *ciliated columnar epithelium* with patches of lymphoid tissues beneath it, causing recurrent inflammation. *Discharge* is mucoid or mucopurulent. Investigations: Discharge study, fistulogram.

Pharyngeal Pouch

It is a protrusion of mucosa through *Killian's dehiscence*, a weak area of the posterior pharyngeal wall between *thyropharyngeus* (*oblique fibres*) and *cricopharyngeus* (*transverse fibres*) of the *inferior constrictor muscle* of the pharynx. *Thyropharyngeus* is supplied by pharyngeal plexus from cranial accessory nerve. *Cricopharyngeus* is supplied by external laryngeal nerve. Imperfect relaxation of the cricopharyngeus

increases the pressure in the pharynx, mainly during swallowing which leads to protrusion of mucosa through the Killian's dehiscence causing pharyngeal pouch (**Fig. 13.24A**). The protrusion is usually towards left.

Stages: (1) Small diverticulum. (2) Large, globular diverticulum causing regurgitation, cough, dysphagia, respiratory infection. (3) Large pouch which is visible in the neck as a globular swelling often tender, smooth and soft. They present with dysphagia, features of respiratory infections like pneumonia and lung abscess, weight loss and cachexia. Pouch may itself get infected and form an abscess. Often the pouch descends downward and enters the superior mediastinum.

Clinical Features

Regurgitation during night or while turning neck, pain, dysphagia, recurrent respiratory infection, swelling in the neck on the left side in posterior triangle which is smooth, soft and tender. Gurgling noise heard while swallowing is typical.

Differential Diagnosis

Branchial cyst; cold abscess; lymph cyst; haemangioma neck.

Investigations

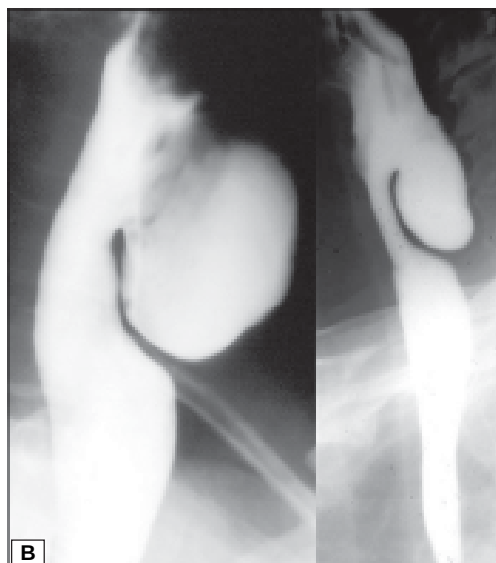
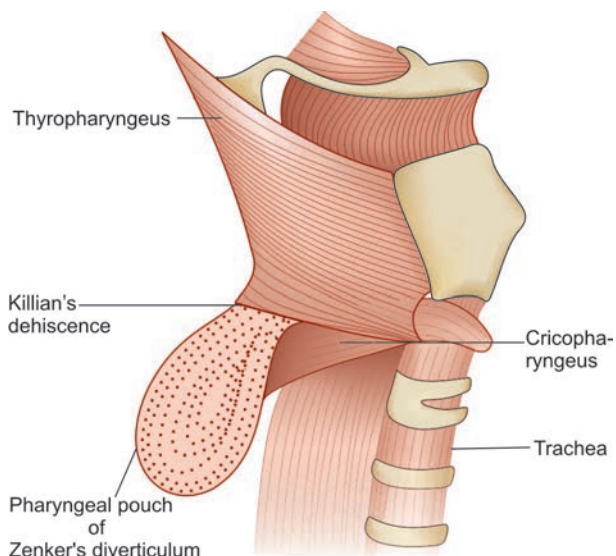
Barium swallow—lateral view shows pharyngeal pouch. Chest X-ray shows pneumonia (**Fig. 13.24B**). CT neck.

Complications

Infection either *mediastinitis* or *lung infection* (pneumonia or lung abscess); pharyngeal fistula; abscess in the neck.

Laryngocele

It is a unilateral narrow necked, air containing diverticulum resulting from herniation of laryngeal mucosa. It is situated in the anterior third of the laryngeal ventricle, between the false cords and thyroid cartilage, herniates through the *thyrohyoid membrane*. It can be external; internal or combined. It occurs in professional *trumpet players*, *glass blowers* and in people

**A**

Figs 13.24A and B: Pharyngeal pouch – anatomical location and barium contrast X-ray picture.

**A****B**

Figs 13.25A and B: Laryngocele becomes prominent and resonant after blowing through nose. X-ray shows air in the neck.

with chronic cough. Swelling is situated in the neck in relation to larynx adjacent to thyrohyoid membrane and is smooth, soft, and resonant becomes more prominent while blowing (Fig. 13.25A). Infection is quite common in the sac of laryngocele, leading to the blockade of opening of the sac causing an abscess. Hoarseness of voice, laryngeal obstruction may

develop. Often there may be repeated discharge of pus into the pharynx. Diagnosis: Clinical features, X-ray neck (Fig. 13.25B), laryngoscopy, CT scan.

Cystic Hygroma (Cavernous Lymphangioma)

It is a cystic swelling due to sequestration of a portion of jugular lymph sac from the lymphatic system, during

the developmental period *in utero*. It presents during birth and so may cause obstructed labour. Occasionally it presents in early infancy.

Sites

Posterior triangle of the neck—commonest site (75%). Eventually may extend upwards in the neck; cheek; axilla; tongue—lymphangiogenetic macroglossia; groin; mediastinum; Often may occur in multiple sites.

Pathology

It contains aggregation of cysts looking like *soap bubbles*. Cysts have *mosaic appearance* with larger cysts near the surface and smaller cysts in the deeper planes. Each cyst contains clear lymph with endothelial lining. Fluid does not coagulate.

Clinical Features

Swelling is *present since birth* in the *posterior triangle of neck* causing obstructed labour; Swelling is smooth, soft, fluctuant (cystic), *compressible, brilliantly trans-illuminant*; Swelling may rapidly increase in size causing *respiratory obstruction—dangerous sign*; It may get infected forming an *abscess* which forms tender, warm, soft swelling. It may cause life threatening septicaemia (**Figs 13.26A to C**).

Complications

Respiratory distress; infection; abscess; septicaemia; surgery itself may cause torrential haemorrhage.

Carotid Body Tumour (Potato Tumour, Chemodectoma, Nonchromaffin Paraganglioma)

It arises from the carotid body, which is located at the bifurcation of the carotid artery.

Cells of the carotid body are sensitive to the changes in pH and temperature of the blood. They are often locally malignant tumours, but in 20% cases spread can occur to the regional lymph nodes. Blood supply to the tumour is from *external carotid artery*. Tumour does not secrete epinephrine or any endocrine substances. They can be *familial*. It is located at the level of hyoid bone deep to anterior edge of the sternomastoid muscle in anterior triangle, vertically placed, round, firm 'potato' like swelling.



Figs 13.26A to C: Cystic hygroma in a newborn. Note the extensive involvement (Courtesy by Dr Manjunath Shetty MS MCh Urology Mangalore).

Clinical Features

Usually unilateral, more common in middle age. Swelling is situated in the carotid region of the neck which is smooth, firm, and pulsatile (due to pulsatile vessel overlying its surface) and moves only side-to-side but not in vertical direction. May present with features of transient ischaemic attacks due to compression over the carotids. Thrill may be felt and bruit may be heard. Often tumour may extend into the cranial cavity along the internal carotid artery as *dumb-bell tumour* (**Fig. 13.27**). *Shamblin classification* of carotid body tumour: Type I—Localised easily resectable (26%); Type II—Adherent, partially surrounding the carotids (46%); Type III—Adherent, carotids encased completely (27%).

Carotid body –

Normal carotid body is 3-5 mm sized flat brownish nodule in the adventitia of common carotid artery.

It consists of chief cells (contains catecholamine granules) and supportive cells.

It gets its nerve supply from glossopharyngeal nerve. These chemoreceptors are sensitive to changes in pH and temperature in the body especially in hypoxia helping in autoregulation of respiration and circulation.

Carotid body hyperplasia can occur in people residing in high altitudes who are exposed to chronic hypoxia. Other chemoreceptors in the body are – *aortic bodies* in the arch of aorta; *glomus jugulare* in the bulb of the internal jugular vein; *glomus intravagale* in relation to ganglion nodosum of the vagus nerve and others like pulmonary (near pulmonary artery) and myocardial (near coronary artery origin) receptors.

Note: Dumb-bell tumours are seen in parotid tumour; spinal cord tumour; carotid body tumour.

Sternomastoid Tumour

It is due to birth injury to the sternomastoid muscle. It is a *misnomer*. It is not a tumour.

Pathogenesis

During child birth injury to sternomastoid muscle causes *haematoma* in the muscle which gets organised to form sternomastoid tumour. It causes congenital torticollis. In congenital torticollis, 1/3rd is due to injury to sternomastoid resulting in sternomastoid tumour; 2/3rd is due to abnormal foetal position *in utero* causing sternomastoid spasm which recovers spontaneously.

Clinical Features

It is seen in infants of 3-4 weeks age. Swelling occurs in the sternomastoid muscle which is smooth, hard, nontender and adherent to the muscle. Chin points towards opposite side and head towards same side (*Scoliosis capitis*). In later age groups it causes *hemifacial atrophy* due to reduced blood supply because of the compression of external carotid artery by sternomastoid tumour (**Fig. 13.28**). Compensatory cervical scoliosis and compensatory squint can also occur.

Differential Diagnosis

Other causes for torticollis.

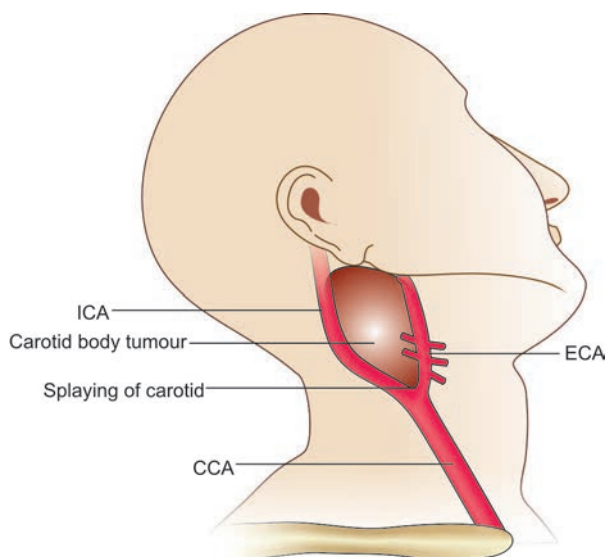


Fig. 13.27 Carotid body tumour.

Investigations

Doppler; Angiogram to see the 'tumour blush'; Widening/splaying of the carotid artery with tumour blush in an angiogram is called as **Lyre sign**. CT scan; No FNAC; No partial excision.

Differential Diagnosis

Carotid aneurysm; soft tissue tumour (Sarcoma); lymph node enlargement; neurofibroma of vagus.



Fig. 13.28: Location of sternomastoid tumour.

Torticollis (Wryneck)

Head is bent to one side with chin pointing towards opposite side. Affected side of face shows mild facial atrophy due to reduced vasculature by restricted movements. Features are - Less arched eyebrow; reduced distance from outer canthus of eye to angle of mouth; flat nose; flat withdrawn cheek.

Causes

Congenital—sternomastoid tumour; *Traumatic* fracture dislocation of cervical spine; *inflammatory* pathology of neck nodes; *spasmodic* due to spasm of same side sternomastoid and posterior neck muscles of opposite side (Fig. 13.29); *compensatory* to scoliosis or ocular causes; *rheumatic* due to exposure to cold; after *burns* contracture.



Fig. 13.29: Torticollis.

Differential Diagnosis for Neck Lymph Node Enlargement

Tuberculous lymphadenitis.
Secondaries in lymph nodes.
HIV infection.
Lymphomas.
Chronic lymphatic leukaemia.
Nonspecific lymphadenitis.
Infectious mononucleosis.
Sarcoidosis.

Tuberculous Lymphadenitis

It is due to *Mycobacterium tuberculosis* infection. Infection is often from tonsils; occasionally from lungs. Tuberculosis may be present in cervical spine. It is common in neck nodes; more often in jugulodigastric nodes (54%); posterior triangle nodes (22%). It can occur in nodes in axilla, para-aortic region, mediastinum, mesentery, iliac region and groin. It may be associated with lymphoma or HIV. It is more common in HIV patients. In tuberculous lymphadenitis – there are *five stages of formation* – stage of lymphadenitis; stage of matting; stage of cold abscess formation; stage of collar stud abscess formation and stage of sinus formation (Fig. 13.30). Often fibrosis and calcification can occur. It contains caseating material with *epithelioid* and *Langerhans' giant cells*.

Types

Caseating: It is commonest type (80%). It shows peradenitis with *matting*, forms cold abscess, collar stud abscess and sinus. It is often resistant to drug therapy. Body resistance is not adequate here. *Hyperplastic* type—is 20% common; discrete, firm nodes are common; shows good response to drugs with better host immunity. Complications are less.

Features

Firm, *matted*, nontender, mobile or fixed swellings in neck often bilateral are the presentations. Tonsils may show tubercles on examination; chronic *midline* retropharyngeal tuberculous abscess may be evident. Lymphoma, secondaries in nodes, nonspecific lymphadenitis, and chronic lymphatic leukaemia are the differential diagnosis.

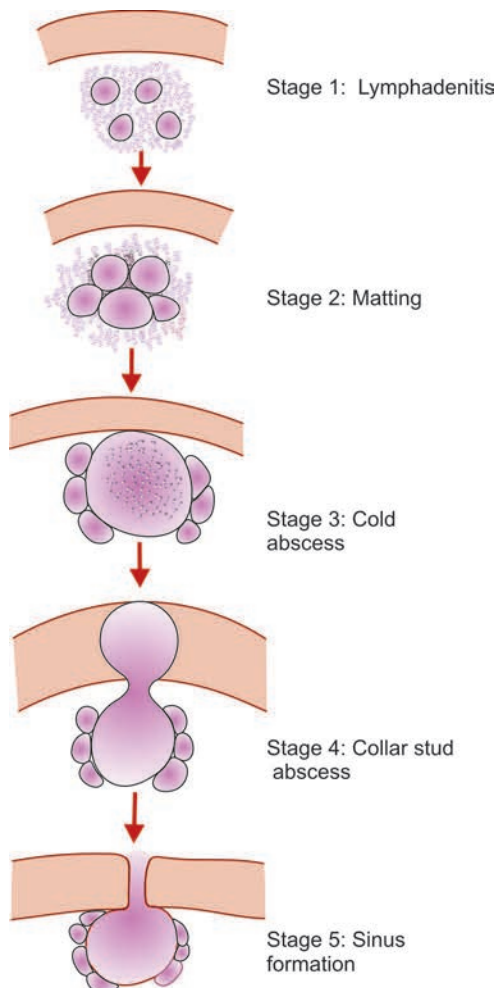


Fig. 13.30: Staging of tuberculous lymphadenitis.

Cold Abscess

It is a complication of tubercular disease. It is commonly observed in neck in relation to caseating tuberculous cervical lymphadenitis. It can occur in relation to spine, like psoas abscess, paraspinal region or any other area. Cold abscess does not show any signs of acute inflammation. It will be soft, smooth, nontender well localised swelling. Relevant lymph nodes, oral cavity/tonsils, cervical/thoracic spines, lungs should be examined. It is well localised, smooth, soft, fluctuant, nontransilluminating swelling with free skin in front (**Figs 13.31 to 13.34**). Branchial cyst,



Fig. 13.31: Tuberculous lymphadenitis—neck forming cold abscess.



Fig. 13.32: Sinus in the neck due to tuberculosis.



Fig. 13.33: Tuberculous ulcer in the neck which is undermined (Courtesy by Dr Balasaraswathy MD DNB, Dermatologist, Mangalore).



Fig. 13.34: Cold abscess forming collar stud abscess in a young boy.

lymph cyst, suppurated lymph node are the *differential diagnosis*.

Investigations are—ESR, chest X-ray, FNAC shows epithelioid cells and Langerhans giant cells, X-ray spine.

Complications are—Sinus formation, systemic spread, secondary infection, resistance tuberculosis.

Cold Abscess

Deep to deep fascia

No evidence of signs of inflammation

Not warm, nontender, smooth, soft and fluctuant, non-transilluminating

Not adherent to skin (skin is free); no redness

Contains cheesy caseating material

It is seen in caseating tuberculous lymphadenitis due to caseation necrosis

It may form collar stud abscess and later sinus

FNAC, AFB, culture are useful investigations

Differential diagnosis are branchial cyst, lymph cyst

Treated by - Antituberculous drugs; 'Zigzag' aspiration by wide bore needle on nondependent area to prevent sinus formation; Drainage using nondependent incision; later closure of the wound without placing a drain.

Secondaries in Neck Lymph Nodes

Levels in Neck Nodes (Memorial Sloan—Kettering Cancer Center Leveling of Neck Nodes)

Level I-Submandibular lymph nodes and submental lymph nodes. Level II-Lymph nodes in upper deep cervical region (It extends from base of skull to hyoid bone and from lateral margin of sternothyroid to

posterior margin of sternomastoid muscle). Level III-Lymph nodes in middle cervical region (From hyoid bone to omohyoid muscle or cricothyroid membrane). Level IV-Lymph nodes in lower cervical region (From omohyoid muscle to clavicle). Level V-Lymph nodes in posterior triangle including supraclavicular region. Level VI-Lymph nodes in the midline neck—pretracheal and prelaryngeal. Level VII-Lymph nodes in the mediastinum. *Note:* Level II and V are now subdivided into Level IIa/Level IIb and Level Va/Level Vb; depending whether these nodes are above the level (Level IIb/Level Va) of the spinal accessory nerve or below (Level IIa/Level Vb). *Note:* Retropharyngeal nodes, facial nodes, post-auricular nodes are not included in these levels.

Common Sites of Primary

Oral cavity, tongue, tonsils; salivary glands; pharynx—nasopharynx; larynx; oesophagus; lungs; GIT; thyroid.

It is commonly from squamous cell carcinoma, but can be from adenocarcinoma, or melanoma. Squamous cell carcinoma is mainly from oral cavity, pharynx. Adenocarcinoma is usually from GIT, commonly involving left supraclavicular lymphnodes (**Figs 13.35A and B**).

Features of Secondaries in Neck

Presents as swellings with nodular surface, hard, often fixed when it is advanced. Secondaries from *papillary carcinoma of thyroid* can be soft, cystic and contains brownish black fluid. Secondaries can infiltrate into carotids, sternomastoid, posterior vertebral muscles, spinal accessory nerve (shrugging of shoulder is affected), hypoglossal nerve (tongue deviates towards the same side), cervical sympathetic chain (*Horner's syndrome*). Secondaries spread into adjacent soft tissues and also to the skin causing fungation and ulceration. Often because of tumour necrosis, softer area develops in the hard node. In advanced cases tumour may infiltrate into the major vessels like carotids, or branches of external carotid artery causing torrential haemorrhage. Dysphagia, dyspnoea, haemoptysis, hoarseness of voice, ear pain, and deafness are other features seen depending on the primary site location.



Figs 13.35A and B: Different types of secondaries in neck – hard nodular; fungating advanced. Involvement of platysma can be made out as a band.

Types of Secondaries in the Neck

1. **Secondaries in the neck with known primary:** Here secondaries are present in the neck and primary has been identified clinically in the oral cavity, pharynx, larynx, thyroid, or other areas. Biopsy from the primary and FNAC from the secondaries has to be taken.
2. **Secondaries in the neck with clinically unidentified primary:** Hard, neck lymph nodes are the secondaries, but primary has not been identified clinically. FNAC of the neck node has to be done and

secondaries have to be confirmed. Then search for the primary has to be done by various investigations. They are Nasopharyngoscopy; Laryngoscopy; Oesophagoscopy; Bronchoscopy; *Blind biopsies* are taken from the fossa of Rosenmuller, lateral wall of pharynx, pyriform fossa, larynx; FNAC of thyroid and suspected areas are done; CT scan.

3. **Secondaries in the neck with an occult primary (70% in jugulodigastric nodes):** Here secondaries in the neck lymph nodes are confirmed by FNAC, but primary has not been revealed by any available investigations. When all the investigations mentioned above do not show any evidence of primary, then only it is called as *occult primary*. Primary tumour is not identified at the time when definitive therapy is started. Initially the secondaries in the neck are treated by *radical neck dissection*, then *regular follow up* is done (at three monthly intervals) until the primary reveals. Once primary is revealed it is *confirmed by biopsy* and treated accordingly, either by *curative radiotherapy* or by *wide excision* depending on location of revealed primary. This type is usually less aggressive and has got better prognosis

Differential Diagnosis

(1) Lymphomas. (2) Tuberculous lymphadenitis. (3) Non-specific lymphadenitis. (4) *Primary branchio-genic carcinoma*. *Reasons for primary lesion being occult:* Primary being too small to be detected; possibility of immunological spontaneous regression of primary and inability of the present diagnostic tools to detect the primary. FNAC is the tool to confirm the occult primary. If FNAC is inconclusive, only then open biopsy (incision/excision) is done to confirm. Open biopsy helps in high suspects of lymphomas or poorly differentiated carcinomas. It facilitates tissue study, immunohistochemistry, and special stains. Many studies prove that risk of seedling, survival and prognosis will not alter by open biopsy. But at present it is proposed only when FNAC fails or special methods are mandatory to type the disease. Immunoperoxidase staining can be done in FNAC specimen or formalin fixed paraffin tissue using monoclonal or polyclonal antibodies. Immunoperoxidase is the most commonly used tool. It is mainly useful in lymphomas/neuroendocrine tumours. Electron microscopy is superior to immunohistochemistry as ultrastructure details can be

assessed. But it is costly. Chromosomal analysis for tumour specific genes is used in B, T and germ cell lymphomas.

Occult primary sites which can cause secondaries in neck

Fossa of Rosenmuller
Lateral wall of pharynx
Posterior third of the tongue
Thyroid
Paranasal sinuses
Bronchus
Oesophagus

Nodal staging in secondaries

N0—nodes not detected
N1—single node same side < 3 cm
N2a—single node same side 3-6 cm
N2b—multiple nodes same side < 6 cm
N2c—bilateral/contralateral nodes < 6 cm
N3—node > 6 cm

Hoarseness—carcinoma larynx, thyroid

Dysphagia—carcinoma posterior 1/3rd of the tongue, pharynx, oesophagus

Haemoptysis, cough, dyspnoea—carcinoma lung

Ear pain, deafness—nasopharyngeal carcinoma

Spinal accessory nerve—Shrugging of shoulder is difficult

Hypoglossal nerve—Tongue deviates to same side with wasting

Sympathetic chain—Horner's syndrome with miosis, anhidrosis, upper eyelid droop (pseudoptosis), enophthalmos, loss of spinociliary reflex.

Rule of 7 in the neck

- 7 days—inflammation
- 7 months—neoplasm
- 7 years—congenital defect

Note: The Rule of 7 provides a probable diagnosis of the neck mass based on the average duration of the patient's symptoms.

Retrosphenoid syndrome/Jacob syndrome is involvement of 6th cranial nerve mainly; 2nd to 6th cranial nerves by neoplastic secondaries causing unilateral ophthalmoplegia, pain, ptosis, trigeminal neuralgia, unilateral weakness of muscles.

Rule of 80 in the neck

80% of nonthyroid neck masses are neoplastic
80% of neoplastic neck masses are seen in males
80% of neoplastic neck masses are malignant
80% malignant neck masses are metastatic
80% of metastatic neck masses are having primary above the clavicle

Primary Branchiogenic Carcinoma

It is rare but important. It is arising from remnants of branchial cleft. It is diagnosed by method of exclusion. It is probably a final histological diagnosis.

Ludwig's Angina

It is an inflammatory oedema of submandibular region and floor of the mouth, commonly due to streptococcal infection. It causes diffuse swelling and brawny oedema of the submandibular region. It is common in severely ill and in advanced malignancy, causing *trismus*, *laryngeal oedema*. Extension of infection into *parapharyngeal space* may lead to dreaded *internal jugular vein thrombosis*. As the infection is deep to the deep fascia in a closed fascial plane, it spreads very fast causing dangerous complications (**Fig. 13.36**).



Fig. 13.36: Abscess in submandibular region.

Clinical Features

Fever, toxicity, diffuse swelling, dysphagia, dyspnoea, trismus, intraoral oedema, brawny submandibular swelling, and putrid halitosis.

Complications

Laryngeal oedema and respiratory distress may require tracheostomy; septicaemia; extension of infection into parapharyngeal space.

Parapharyngeal Abscess

It is *infection of pharyngomaxillary space*. This is a cone shaped space with base formed by base of the

skull and apex by the greater cornu of hyoid bone; medial wall by the superior constrictor; lateral wall by the internal pterygoid, angle of mandible and submandibular salivary gland. Usually infection arises from the tonsils after tonsillectomy and from the submandibular space.

Clinical Features

It causes diffuse swelling in the upper neck, trismus, fever, toxicity.

Complications

Thrombosis of internal jugular vein; Erosion into the internal carotid artery causing torrential bleeding; Septicaemia.

Retropharyngeal Abscess

Surgical Anatomy

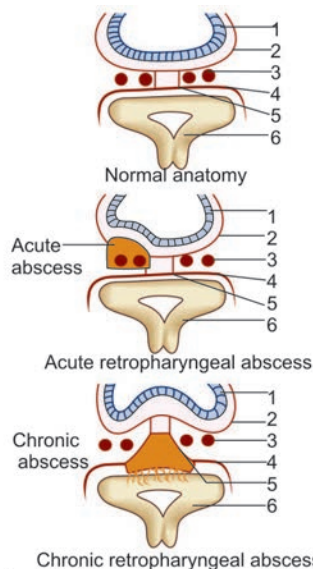
The wall of the pharynx has got 5 layers. Mucosa, submucosa, pharyngobasilar fascia, muscular layer (contains 3 constrictors and stylo, salpingo, palatopharyngeus muscles) and buccopharyngeal fascia covers outer part of constrictors and extends over buccinator. Buccopharyngeal fascia is adherent to prevertebral fascia posteriorly in the midline. Retropharyngeal lymph nodes are located between buccopharyngeal fascia and prevertebral fascia in paramedian (eccentric) position (Not midline) (**Fig. 13.37A**).

Types—Acute; Chronic

Acute retropharyngeal abscess: It is infection and suppuration of retropharyngeal lymph nodes due to staphylococci or streptococci organisms; commonly from tonsils or pharynx; common in infants and children.

Clinical features: It presents as *lateral (paramedian, eccentric) smooth, tender swelling* in the pharynx with dysphagia, dyspnoea, cough, toxic features and neck rigidity. Diagnosis is obvious on proper clinical examination. It is drained intraorally under general anaesthesia.

Chronic retropharyngeal abscess: It is invariably due to tuberculosis of cervical spine. Abscess is located



1. Pharyngeal wall 2. Buccopharyngeal fascia 3. Retropharyngeal LN 4. Prevertebral fascia 5. Median septum 6. Vertebra

A



B

Figs 13.37A and B: Note acute and chronic retropharyngeal abscess. Normal anatomy is also shown. Acute is eccentric and is due to suppuration of retropharyngeal lymph nodes. Chronic is central, midline and is due to tuberculosis of the cervical vertebra. X-ray picture showing retropharyngeal abscess due to tuberculosis.

in the *midline behind the prevertebral fascia*. There is destruction of the body of the vertebra due to tuberculosis.

Clinical features: It is *midline swelling in the posterior pharyngeal wall*, which is smooth and nontender. Features of tuberculosis of cervical spine will be observed. Often abscess may point in the neck in relation to sternomastoid. Neurological manifestations may occur in severe disease.

Investigations: X-ray spine, chest X-ray, ESR, MRI of cervical spine are essential investigations. Drainage of the abscess should be done *through neck approach (never intraoral approach)* (**Fig. 13.37B**).

Thoracic Outlet Syndrome (TOS)

It is syndrome complex due to compression of neurovascular bundle in the thoracic outlet. *Thoracic outlet has got two main spaces—Scalene triangle* which is bound by scalenus anterior, scalenus medius and first rib and contains subclavian artery and brachial plexus; *Costoclavicular space* which is bound by clavicle, first rib, costoclavicular ligament and scalenus medius and contains subclavian artery, vein and brachial plexus.

Causes

Cervical rib; long C7 transverse process; anomalous insertion of scalene muscles; scalene muscle hypertrophy; scalene minimus; abnormal bands and ligaments; fracture clavicle or first rib; exostosis; tumours in the region; brachial plexus trauma and diseases.

Differential Diagnosis of TOS

Carpal tunnel syndrome; cervical spondylosis; spinal canal tumours; shoulder myositis; angina; Raynaud's disease; spinal stenosis; ulnar nerve compression; Epicondylitis.

Clinical Features

Neurological—Paresthesia; pain in shoulder, arm, forearm and fingers; occipital headache as referred pain from tight scalene muscles; weakness in forearm, hand.

Vascular—Claudication, ischaemic ulcers, gangrene.

Signs

Scalene muscle tenderness; pulsatile swelling in supraclavicular region with thrill and bruit (25%); bony mass above clavicle; Adson's test positive; Roos test positive; Elevated arm stress test (EAST) positive; costoclavicular compression maneuver and hyperabduction maneuver positive; poor capillary refilling; absence or feeble pulse (*Please refer Chapter 5: Examination of Arterial Diseases for details of tests*).

Cervical Rib

Definition

It is an extension of transverse process of C7 vertebra more than 2.5 cm (normal). Syndrome caused by it is called as *cervical rib syndrome, thoracic inlet syndrome, thoracic outlet syndrome; scalene syndrome*. It is 0.46% common, common in females, more frequently on right side. It can be *unilateral or bilateral*; can be *asymptomatic or symptomatic*.

Types

- (1) **Complete bony:** Cervical rib is radio-opaque, anteriorly ends over the first rib or manubrium.
- (2) **Fibrous:** Cannot be demonstrated radiologically.
- (3) **Combined:** Partly bony partly fibrous.
- (4) **Partial bony:** With free end expanding as bony mass.

Pathology

Cervical rib narrows the scalene triangle (bounded by scalenus anterior, scalenus medius and first thoracic rib below) → compression of subclavian artery; C₈ and T₁ nerve roots due to cervical rib → angulation of subclavian artery → causes constriction of artery at the site where artery crosses the cervical rib → '*Eddie's current*' created in the blood flow causes sudden release of pressure distal to the narrowing → *Post-stenotic dilatation* due to spasm of vasa vasorum of localised segment of the artery → *Venturi phenomenon* → stasis of blood → *Thrombosis* → *Embolus* → Features of ischaemia in the hand and forearm. Later digital gangrene develops (**Fig. 13.38A**). Compression of C₈ and T₁ causes tingling and numbness along its distribution, i.e. in the little finger, medial side of hand and forearm. Rarely thrombus may extend proximally into the subclavian artery causing vertebral basilar insufficiency. *Paget-Schrotter syndrome* is subclavian vein compression by cervical rib. It is rare.

Clinical Features

Majority of patients are asymptomatic.

Neurological features (Most common presentation):

Is due to compression of T₁ and C₈ causing sensory (tingling and numbness in the little finger, medial side of hand and forearm); motor (wasting of thenar and hypothenar eminence with often claw hand and loss of power of the hand); vasomotor with excessive sweating of the hand.

Vascular manifestations (More problematic presentation):

Pain is due to ischaemia in the muscle. It is more during work, exercise and is relieved by rest. **Roos test** is positive (i.e. raising the arm above the shoulder. The side where cervical rib is present, patient cannot continue and so drops the hand down). **EAST-Elevated Arm Stress Test (Modified Roos test)**: Arm is elevated above the shoulder, with elbow stretched fully. Rapid movements of fingers will cause fatigue on the side where cervical rib is present. **Adson's test**: The hand is raised above after feeling the radial pulse. The patient is asked to take a deep inspiration and turn the head to the same side. Any change in pulse, i.e. either becoming feeble or absent is noted. **Modified Adson's test** is same as Adson's, but neck is turned towards the opposite side. Wasting of thenar, hypothenar and forearm muscles are evident. Often **digital gangrene** may be observed. Limb is colder and pallor than the opposite side.

Features in the neck: (a) Hard, fixed, bony mass in the supraclavicular region. (b) *Palpable thrill* above the clavicle in the subclavian artery. (c) *Bruit* on auscultation.

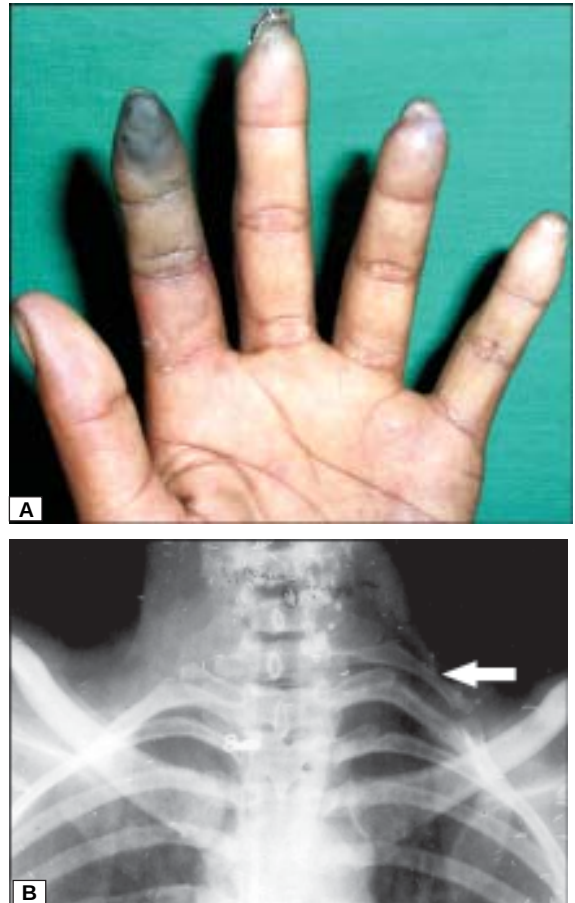
Differential Diagnosis

(1) Cervical spondylosis—X-ray neck—lateral view should be taken to differentiate. (2) Carpal tunnel syndrome. (3) Tumours or swellings compressing over the vessel or nerves in the neck. (4) Other causes of digital gangrene like Raynaud's, atherosclerosis, diabetes, collagen diseases, embolism. (5) Syringomyelia, motor neuron disease. (6) Pancoast tumour.

Investigations

Chest X-ray PA-view and lateral view including neck-only bony rib (radiopaque) can be identified. Nerve

conduction studies to confirm neurological compression and also to rule out carpal tunnel syndrome or cervical spondylosis. Arterial Doppler of subclavian artery and of the upper limb. Subclavian angiogram is done to look for vascular involvement (**Fig. 13.38B**).



Figs 13.38A and B: Cervical rib causing gangrene of digits. X-ray showing cervical rib.

Subhyoid Bursitis

Subhyoid bursa is space between posterior surface of the body of hyoid bone and thyrohyoid membrane. It lessens friction between these two structures during swallowing. Due to constant friction inflammatory fluid collects in the bursa leading to bursitis, which presents like a *horizontally placed* midline swelling between lower part of the hyoid bone and thyrohyoid membrane.

Features

Smooth, soft, cystic, fluctuant, non-transilluminant swelling which moves upwards with deglutition but *not while protruding the tongue out*. It should be differentiated from thyroglossal cyst and pretracheal lymph nodes. It contains turbid fluid which often may get infected to make swelling tender or to form an abscess (**Fig. 13.39**).

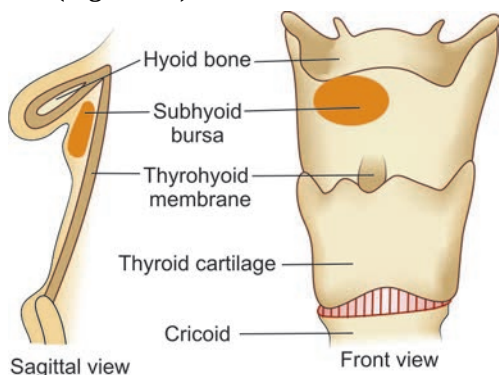


Fig. 13.39: Sagittal and front view showing location and relation of subhyoid bursa.

Anatomy of Lymphatics of the Head and Neck

Waldeyer's lymphatic ring (Inner) (Fig. 13.40): It consists of adenoids above, lingual tonsils below and two palatine tonsils laterally one on each side.

Outer circular chain of nodes (outer Waldeyer ring): Occipital, post-auricular, preauricular, parotid, facial, submandibular, submental, superficial cervical and anterior cervical.

Facial nodes: *Superficial*—Upper-infraorbital; Middle-buccinator; Lower-supramandibular; *Deep groups*—in relation to pterygoids;

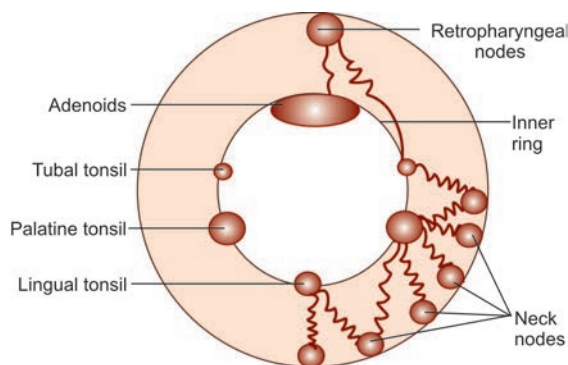


Fig. 13.40: Waldeyer ring

Submandibular lymph nodes drain: The side of the nose; cheek; angle of the mouth; entire upper lip; outer part of the lower lip; the gums; side of the tongue.

Submental lymph nodes drain: From the central part of the lower lip, floor of the mouth and apex of the tongue.

Superficial cervical nodes: They lie on outer surface of the sternomastoid around the external jugular vein. They drain the parotid region and lower part of the ear.

Deep cervical lymph nodes: Upper deep cervical lymph nodes—*jugulodigastric nodes*; Lower deep cervical lymph nodes—*jugulo-omohyoid nodes*; middle deep nodes. They drain from that half of head and neck and finally form a *jugular lymph trunk* from lower deep cervical to join thoracic duct on the left side and junction of right subclavian and right jugular vein on right side.

14

Examination of Thyroid

History taking begins with:

1. **Name:** It is first and basic requirement.
2. **Address:** Knowing the residential place may be important in certain types of goitres. – endemic goitre due to iodine deficiency is common in interior regions, mountainous areas like Vindhya, Himalayas. Goitre is more common in south India than north India. It is also common in Middle East and European countries, North America, Bulgaria near river Struma which eventually reaches Aegean Sea. Follicular and anaplastic carcinoma may be more common in iodine deficiency areas but papillary carcinoma is not related to iodine deficiency. Chalk or limestone producing areas like Southern Ireland and Derbyshire are goitrogenic areas as calcium is goitrogenic (**Fig. 14.1**).
3. **Occupation:** Not much related to thyroid diseases.
4. **Age:** Simple goitre is often seen in girls during puberty. Goitre due to dys hormonogenesis occurs in younger age group. Physiological goitre occurs when there is increased metabolic demand of the hormone like in puberty, pregnancy. Solitary nodule, colloid goitre, papillary carcinoma and primary thyrotoxicosis are seen between 20-40 years. Multinodular goitre, follicular carcinoma and Hashimoto's thyroiditis are seen in middle aged women.

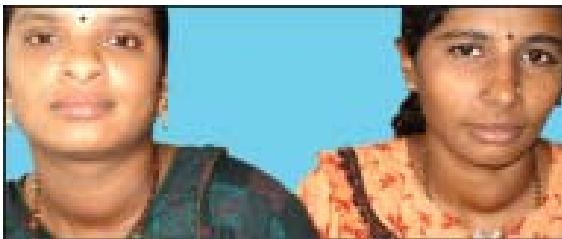


Fig. 14.1: Goitre may occur in many family members. Two siblings presented with goitre.

5. **Sex:** Most of the thyroid diseases like hyperthyroidism (8:1), hypothyroidism, goitres, neoplasms (3:1) are commonly seen in *females*.

Chief Complaints

Swelling in front of the neck and its duration; *Pain* in the swelling and its duration; *Hoarseness of voice* due to recurrent laryngeal nerve palsy; *Difficulty in swallowing* or breathing; *Tremor* in the hands; *Generalised weakness*; *Palpitation*; *Loss of significant weight*.

History

History of Present Illness

Swelling

Its duration, mode of onset whether sudden or insidious in nature should be asked. Origin of the swelling, its progress whether gradual (benign) or rapidly progressive (malignancy) or recent rapid increase in a pre-existing swelling (benign turning into malignancy) or sudden rapid increase may be seen in haemorrhage. Haemorrhage or malignant transformation (follicular carcinoma) can occur in a preexisting multinodular goitre. Thyroglossal cyst may be present since childhood. Swelling may be single/multiple or occupying one lobe, or both lobes or isthmus. Most of the goitres, solitary nodule, multinodular goitre are slow growing swellings. Anaplastic carcinoma, follicular carcinoma, medullary carcinoma are rapidly growing tumours. Papillary carcinoma which is the commonest thyroid malignancy, even though malignant is a slow growing tumour often for few years.

Note: Any thyroid of any size or any duration or any consistency or in any age group can be malignant unless proved otherwise.

Pain

Its duration, character like dull aching/pricking, site of pain, radiation, factors which alters the pain should be asked for. Usually goitres are painless. Thyroiditis may be painful. Malignancy is initially painless but later becomes painful. Infiltration into surrounding structures (nerves)/necrosis/haemorrhage makes it painful and tender. Anaplastic carcinoma commonly infiltrates into nerves to cause pain.

Pressure Symptoms

Dysphagia (oesophageal compression), dyspnoea (tracheal compression), stridor (infiltration into trachea), hoarseness of voice (recurrent laryngeal nerve compression) and Horner's syndrome (infiltration of cervical sympathetic chain – ptosis, loss of sweating, in face same side, miosis and enophthalmos). Their duration, onset and progression should be asked.

Features of Toxicity

Increased appetite/loss of weight/diarrhoea/chest pain aggravated by exercise/palpitation/amenorrhoea/irritability/nervousness/sleeplessness (insomnia)/hand tremors/increased sweating/cold preference/heat intolerance/proximal muscle weakness in the thigh or arm like fatigue on getting down steps or lifting weight using arms (myopathy)—due to difficulty in isometric contraction and increased muscle metabolism/wasting of muscles/visual disturbances with bulging of the eyes (exophthalmos). Usually in primary thyrotoxicosis, symptoms appear first which are more severe than secondary type; later diffuse thyroid swelling appears in the neck. Here often swelling in the neck may not be present or may not be obvious. In secondary thyrotoxicosis obvious swelling appears first which is nodular later symptoms of thyrotoxicosis appear which are less severe initially compared to primary thyrotoxicosis; but symptoms gradually become more severe. Neurological and eye signs are more common in primary thyrotoxicosis.

Features of hypothyroidism/myxoedema: Muscle weakness/lethargy/weight *gain*/poor appetite/facial swelling/cold intolerance/menorrhagia/constipation/superciliary madarosis in lateral half of the eyebrows/loss of hairs in scalp/change in voice due to vocal

cord oedema/dry skin/muscle fatigue/lethargy/less memory/sleepiness. *Myxoedema crisis* may develop with acute exacerbation of features.

Past History

History of irradiation should be asked for carcinoma thyroid. Irradiation to head and neck region may have been given for benign lesions like adenoids, tonsillitis, thymus, acne vulgaris or haemangiomas or malignancy in younger age groups like for lymphomas. Chernobyl nuclear disaster in Ukraine in 1986 caused increased incidence of papillary carcinoma of thyroid in children in that area. Previous history of having thyroglossal cyst must be noted which might have been infected causing fistula either due to spontaneous burst or after surgical drainage of the infected cyst. Previous surgery for thyroid in recurrent thyroid swelling or earlier surgery for thyroglossal cyst in case of thyroglossal fistula should be asked for.

Personal History

History of smoking, alcohol intake or any drugs which may cause alteration in thyroid function should be asked. History of any drug intake like patient may be on thyroxine or on antithyroid drugs or beta blockers or other drugs like lithium, PAS or sulphonylureas which may alter the thyroid should be noted. Dietary habits should be asked. Vegetables belonging to Brassica family like cabbage, kale and rape are goitrogens. Type of salt used in the family iodized/home rock salt is also important.

Family History

Dyshormonogenesis, medullary carcinoma of thyroid can be familial (MEN syndrome). Endemic goitre and Grave's disease can occur in families. Altered thyroid function may be the cause for infertility.

Menstrual History

History of menarche/menopause; duration of menstruation, history suggestive of menorrhagia, amenorrhoea, oligomenorrhoea, etc. should be asked for. Hyperthyroidism can cause amenorrhoea; hypothyroidism may cause menorrhagia.

Treatment History

History of undergoing investigations or treatment relevant to thyroid disease should be asked for. Patient may be taking L thyroxine (as once a day – small tablet) or may be taking antithyroid drugs like carbimazole or propylthiouracil usually three times a day for hyperthyroidism. History should be detailed whether patient is becoming better after therapy or not (drug response). Often patient may be taking drugs like PAS or sulphonylureas which are goitrogenic. History of intake of drugs for other diseases should be mentioned.

General Examination

Like any other long case. Thyrotoxic patient is anxious/thin and undernourished. Obesity is seen in myxoedema. Patient may be cachexic in thyroid carcinoma which is advanced/metastatic. Exophthalmos should be looked for in toxic patient. Irritable/agitated tensed face with eye signs is seen in toxic thyroid. Myxoedema face is typical. It is expression less, *mask-like puffy* face. Patient will be dull with low intelligence (*everything is slow* – walking, talking, moving, thinking, reflex). Hasty – rapid gait is seen in hyperthyroid and slow-lethargic gait in hypothyroidism. **Pulse**—Its character, whether tachycardia, collapsing/Corrigan's or pulsus paradoxus type or ectopic or fibrillation has to be looked for. Pulse rate may be slow in hypothyroidism. Sleeping pulse rate is checked at late night or early morning for three consecutive nights and average is taken. Sedating with diazepam or phenobarbitone to check sleeping pulse rate prior to sleep is a controversial (better to avoid). Sleeping pulse rate is graded as per Crile's grading. Blood pressure may be high in toxic thyroid (**Figs 14.2 and 14.3**).

Crile's grading	Sleeping pulse rate/minute
I	Up to 90
II	90-110
III	> 110

In toxic thyroid, patient will be thin and underweight in spite of patients' good appetite. In hypothyroidism, patient will be obese and overweight. Skin is wet and warm in hyperthyroidism (*moist palm while shaking hands*). Ankle (*Achilles tendon*) reflex is prolonged with delayed relaxation in hypothyroidism and it is shortened and brisk in hyperthyroidism. Both legs and



Fig. 14.2: Palpation of radial pulse for its count, volume, variations should be done in thyroid diseases.



Fig. 14.3: Thyrotoxicosis with exophthalmos.

ankle region in front should be inspected for *pretibial myxoedema*. It is a feature of primary thyrotoxicosis. It is due to deposition of myxomatous tissue (**Fig. 14.4**).

Tremor of the Hands and Tongue

Hand tremors are observed in outstretched hands and fingers. Often small object like pen may be kept to watch the tremor better. Fine tremors are observed in toxic thyroid (**Figs 14.5A to C**). It is due to diffuse irritation of the grey matter. Tongue twitching can be observed by opening the mouth and carefully observing the tongue. Tongue should be *within* oral cavity to check the tremor. Protruded tongue causes fasciculation of intrinsic muscles of tongue which mimic tongue tremor.



Fig. 14.4: Pretibial myxoedema is seen in primary thyrotoxicosis.

Assessment of voice change:

- Pitch of the voice—whether raised/lowered or pitch locked
- Breath support during speaking whether adequate or not
- Ability to alter the rapidity of speech—slow/fast/medium
- Altered laryngeal and neck muscle tension

Indirect laryngoscopy—with tongue pulled out using gauze, warmed ILS is passed into the oral cavity to see vocal cords. Patient is asked to say 'e' to see the vocal cord movements

Local Examination

Inspection

Thyroid is the only endocrine gland which is properly clinically accessible; only gland can be involved in all age groups; only gland where malignant tumours are mostly nonfunctioning.

Inspectory findings of the swelling should include (Figs 14.6A and B): Its location/size (both vertical and



Figs 14.5A to C: Tremor of the outstretched hands and tongue (within) should be checked properly in toxic thyroid.



Figs 14.6A and B: Simple goitre. Inspection of goitre is very important.

horizontal dimensions of each lobe and isthmus or if it presents as a single mass dimension of the single swelling)/shape (butterfly shape if both lobes are involved)/extent (from posterior border of sternomastoid laterally to midline in single lobe enlargement of gland or from one side to opposite sternomastoid if both lobes are enlarged)/ upper extent is usually up to thyroid cartilage/*lower margin may or may not be clearly visible* or may be visible during deglutition/**upwards movement with deglutition** (thyroid moves upwards during deglutition due to attachment of the condensed vascular pretracheal fascia (*Berry's ligament*) above (**Fig. 14.7**), medially and behind to cricoid cartilage and this *pretracheal fascia* is also attached to larynx, thyrocricoid cartilage → thyrohyoid membrane → hyoid with *inferior constrictor muscle which moves upwards during deglutition*)/scar or



Fig. 14.7: Thyroid moves upwards with deglutition. Often it is better to give a glass of water to the patient to drink.

dilated veins (in toxic goitre, carcinoma thyroid, venous compression, retrosternal goitre) or pigmentation on the skin over the swelling/pulsation over the swelling (toxicity, malignancy)/surface on inspection (smooth or nodular). *Pizzillo's method* of inspection is done in obese short necked individuals by pushing the head backwards against clasped hands placed over the occiput.

Surface is smooth in physiological goitre, primary toxic goitre and Hashimoto's thyroiditis. It is nodular in multinodular goitre. In malignancy it can be smooth or nodular. Thyroglossal cyst, subhyoid bursa, prelaryngeal or pretracheal lymph nodes also move with deglutition (**Figs 14.8 to 14.10A**).

Swellings which move upwards with deglutition

Thyroid swelling
Subhyoid bursa
Thyroglossal cyst
Pretracheal / prelaryngeal lymph nodes
Swelling from larynx / trachea

In some occasions whether swelling moves while protruding the tongue or not should be looked for. Thyroglossal cyst moves upwards with protrusion of tongue. Patient is asked to open the mouth and then swelling/cyst is held firmly. Now patient is asked to protrude the tongue out to feel an upward movement of the swelling with a typical '**tug**' like feeling in the swelling on inspection. Thyroglossal fistula presents as a withdrawn opening in the *midline* below



Fig. 14.8: Diffuse toxic goitre. Surface of thyroid here is smooth. Exophthalmos is also present.



Fig. 14.10A: Recurrent nodule thyroid. Patient has undergone thyroidectomy once earlier. Note the scar in the neck.



Fig. 14.9: Large carcinoma of thyroid which is vascular. Note the dilated veins.



Fig. 14.10B: Thyroglossal cyst.

the hyoid bone having a *crescentic fold of skin* (**Fig. 14.10B**).

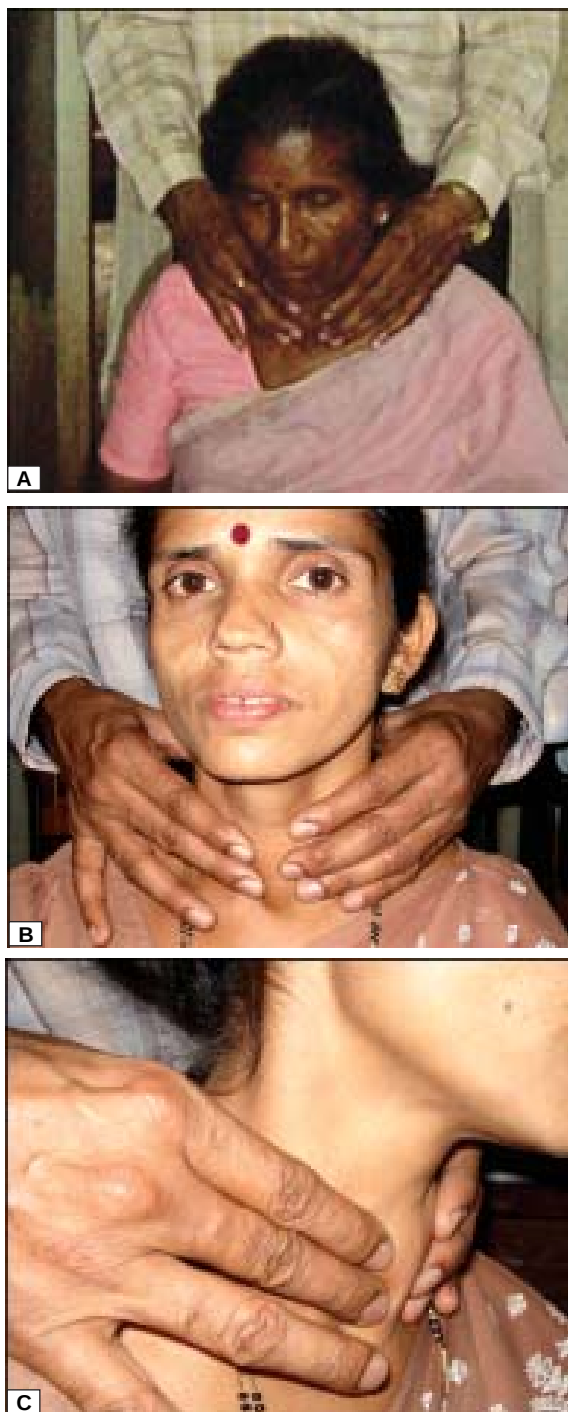
Occasions wherein thyroid swelling may not move upwards with deglutition-

Anaplastic carcinoma thyroid – often
 Carcinoma thyroid with extensive local infiltration into soft tissues, trachea / larynx and posterior muscles
 Intrathoracic retrosternal extension with infiltration / impaction
 Riedel's thyroiditis with encasement of trachea
 Massive thyroid wherein upward movement is difficult to observe and appreciate

Any other swelling in the neck should be looked for like lymph nodes. Lymph nodes are commonly involved in papillary carcinoma of thyroid and occasionally in follicular carcinoma of thyroid.

Palpation

Palpation of thyroid is done from behind with the patient sitting on a stool comfortably and flexing the neck. Both thumbs of the examiner are placed over the back of the neck and fingers of each hand are placed on the respective lateral lobes for palpation (**Figs 14.11A to C**). Isthmus should also be palpated like this. For detailed palpation of one side lateral lobe



Figs 14.11A to C: Palpation of thyroid from behind with patient sitting in a stool comfortably and neck flexed. Careful palpation for nodules should be made.

patient is made to flex the neck towards that side to relax the sternomastoid muscle. But many specific tests are done with examiner standing in front.

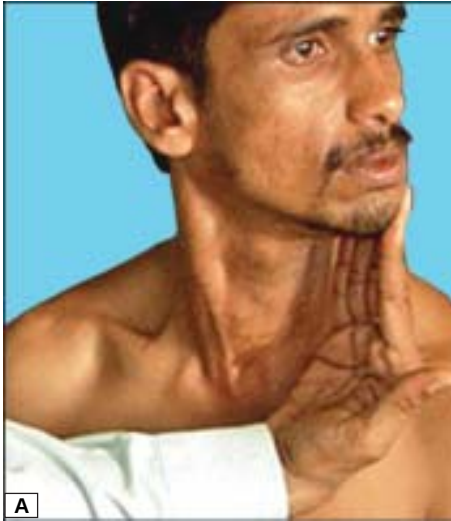
Palpatory findings of the swelling includes: Temperature over swelling (swelling may be warm in toxic thyroid, malignancy, thyroiditis)/tenderness (haemorrhage, thyroiditis, tumour necrosis can cause tenderness)/extent/position/shape/size (should be measured in centimeter both vertically and horizontally)/movement of the swelling upwards with deglutition/surface (smooth or nodular)/consistency (soft or firm or hard or variable and if so different locations of different consistencies should be mentioned)/margin (well defined or diffuse, lower margin which is most important should be specially mentioned)/independent mobility of the swelling/plane of the swelling (it is checked by contracting the sternomastoid muscles by placing examiner's hand under the chin of patient and patient has to flex the neck against resisting hand) (relation of the single side gland to sternomastoid muscle is checked by contracting the muscle by turning the chin against resistance of the examiner's hand which is placed on the opposite side)/whether skin is free or not. *Pizzillo's method* also can be used for palpation in short neck and obese patients to make nodules more prominent (**Figs 14.12A to 14.14**).

Thyroid swelling moves upwards with deglutition; but does not show independent upward mobility; it shows horizontal mobility along with trachea. A small encapsulated swelling occasionally may show independent free mobility.

Surface is smooth in primary toxic goitre, Hashimoto's thyroiditis; nodular in multinodular goitre. **Consistency** in thyroid swelling is variable in malignancy; it can be soft/firm/hard. It is hard in Riedel's thyroiditis and calcified cyst. It is firm or hard in multinodular goitre. It is soft in colloid goitre, physiological goitre and primary toxic goitre. Tensely cystic swelling can be felt hard; neoplastic solid swelling can be softer—*thyroid paradox*.

Lower margin of the gland/swelling should be checked during deglutition by placing the examiner's index finger horizontally just above the sternum.

Thrill is checked in the upper pole of the gland as superior thyroid artery is superficial and enters the upper pole of gland in front. Thrill signifies toxicity or increased vascularity (**Figs 14.15A and B**).



A



A

Figs 14.12A and B: Contraction of sternomastoid one side/ both sides to confirm that thyroid is deep to deep fascia.

Method of Palpation of Thyroid Gland

Thyroid gland is palpated from behind with patient sitting on a stool with neck partially flexed. Both thumbs of the examiner are kept over the cervical spine and fingers placed in front to feel the gland—both lateral lobes and isthmus for all features.

Crile's Method of Palpation of Gland

It is the palpation of the nodule/swelling in front using the pulp of the thumb when patient is swallowing (**Fig. 14.16**).

Pizzillo's Method

It is the method of inspecting and often palpating the thyroid gland in short necked and obese



Fig. 14.13: Lower border should be assessed in case of thyroid enlargement to rule out possible retrosternal extension.



Fig. 14.14: Skin should be pinched to confirm that swelling is not adherent to skin.



Figs 14.15A and B: Superior pole of thyroid should be palpated for thrill which signifies vascularity.

individuals. Patient is asked to keep her/his clasped hands over the occiput and head is pushed against the hands; gland which becomes prominent will be inspected or palpated from front or behind (**Fig. 14.17**).

Lahey's Method of Examination

It is the method used to palpate any nodules in the posterior part of the gland. It is mainly useful in solitary nodule of thyroid. Examiner should stand in front of the patient. If right lobe is needed to be palpated, left lateral lobe is pushed towards right to make posterior aspect of the right lobe more prominent as gland gets pushed and rotated towards right side. Posterior becomes posterolateral (by rotation of trachea) or



Fig. 14.16: Crile's method of palpation using thumb for any nodules.



Fig. 14.17: Pizzillo's method of examination.

lateral which is felt for any nodules. Posterior aspect of left lobe is palpated by pushing the right lobe towards left side (**Fig. 14.18**).

Kocher's Test

It is the test to check for tracheal compression. Patient is asked to see straight. With fingers and thumb both lateral lobes of the thyroid gland are gently compressed directing posteromedially. If patient develops stridor—Kocher's test is positive. If patient develops no stridor, it means test is negative (**Fig. 14.19**). In a long standing goitre and large goitre, weakening of tracheal



Fig. 14.18: Lahey's test.



Fig. 14.19: Kocher's test.

rings occurs because of constant pressure which gets narrowed/collapsed during *compression*. Trachea is kept patent because of forward traction by goitre itself. But after thyroidectomy lack of support to trachea causes *tracheomalacia*—weakening of the tracheal rings. Such patients need tracheostomy after thyroidectomy. It is usually temporary tracheostomy for 2-3 weeks, by then tracheal rings regain their strength to maintain the patency of the trachea. 'Scabbard trachea' is narrowing of trachea.

Confirmation of Retrosternal Extension

Lower margin of the swelling/goitre is not visible—even on deglutition. Lower margin is not palpable on deglutition. Dilated veins over neck or chest wall may be visible. Normal resonant note over the sternum becomes dull on percussion.

Pemberton's sign: Patient is asked to raise both the arms above the shoulder so as to touch the ears and asked to keep like that for 3 minutes. Patient will develop dilated veins and cyanosis in the neck and upper chest wall, puffiness in face, respiratory distress and rarely dysphagia. It means sign is positive signifying retrosternal extension of goitre (**Figs 14.20 and 14.21**). Dyspnoea can occur at night during lying down or when neck is extended. Rarely recurrent laryngeal nerve palsy can occur.

Retrosternal goitre is defined as having > 50% goitre below the suprasternal notch. Primary is rare – 1%. Primary retrosternal goitre arises from ectopic thyroid tissue from mediastinum. It gets its blood supply from mediastinum itself, not from the neck. And it also is not related to the existing thyroid in the neck. Secondary is common. It is extension from



Fig. 14.20: Pemberton's sign for retrosternal goitre.

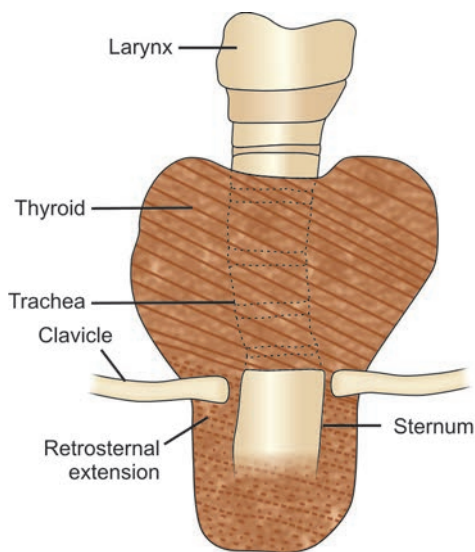


Fig. 14.21: Diagrammatic presentation of retrosternal extension of thyroid.

the enlarged thyroid from the neck. Commonly retrosternal goitre arises from lower pole of a nodular goitre. It is more observed in short necked people. Due to negative intrathoracic pressure nodule gets drawn into the superior mediastinum. Sometimes it may be also ectopic thyroid tissue. Retrosternal goitre may be *substernal* (part of the nodule is palpable in the neck) or *plunging* goitre (intrathoracic goitre is forced into the neck occasionally by increased intrathoracic pressure) or *intrathoracic goitre* with normal neck. It can be toxic/nontoxic nodules/malignant.

Retrosternal goitre is confirmed by CT scan and radioiodine isotope study. It is treated by complete surgical removal usually through neck approach, occasionally through median sternotomy. Radioactive iodine therapy is not used for retrosternal goitre. *Surgical removal should be complete because recurrent retrosternal goitre is very difficult to re-operate. Stridor due to compression of tracheo-bronchial tree by retrosternal goitre is very dangerous because it is often not possible to clear the airway either by intubation or by tracheostomy.*

Position of Trachea

Position of trachea is checked by palpation using three fingers from below. Middle finger is kept just above the

suprasternal space and index and ring fingers are placed over sternal heads of the sternomastoid muscles on each side. Middle finger is run from above downwards along the trachea to feel the position—central or deviated. In solitary nodule or disease of only one lateral lobe trachea will be usually deviated towards opposite side. In enlargement of both lobes trachea will be usually central. Other features are absence of hollowness on the side of the deviation (*trail sign*), on auscultation breath sounds are heard well on the side of the deviation (**Figs 14.22 and 14.23A and B**).

Superior border of the isthmus of the normal thyroid gland is inferior to cricoid cartilage. Isthmus is felt over the tracheal rings below. *Bare tracheal rings* are observed in ectopic thyroid (which means thyroid tissue is not present in normal location) and also in absence of isthmus (rare).

Carotid pulsation should be checked. It is normally felt at the level of the upper border of thyroid cartilage over medial aspect of the sternomastoid muscle on the Chaissagne tubercle (carotid tubercle) on the transverse process of C6 vertebra. It may be deviated posteriorly/laterally in a large goitre. It may be absent in advanced carcinoma thyroid due to infiltration of the carotid sheath by the tumour (*Berry's sign*) (**Fig. 14.24**).

Sympathetic chain in the neck may get involved in locally advanced carcinoma thyroid causing

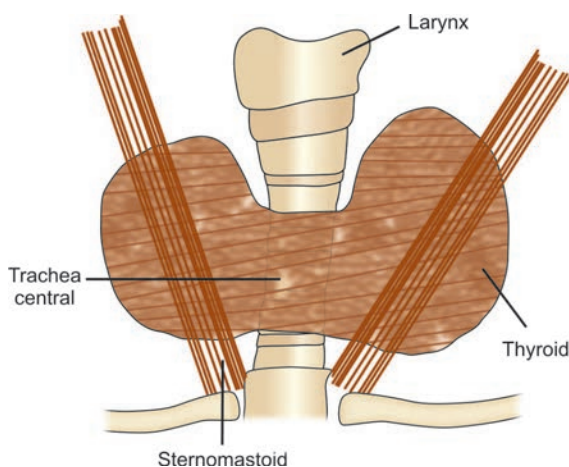
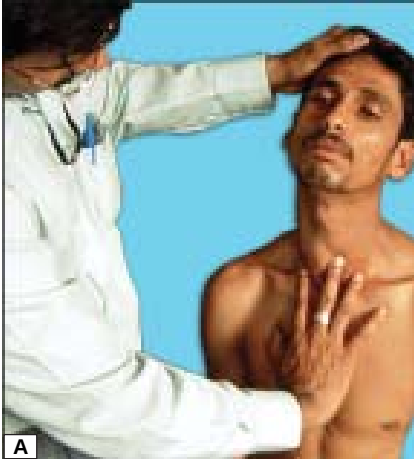


Fig. 14.22: Trachea in central position. It is central when both lateral lobes are enlarged. It is deviated to opposite side in solitary nodule thyroid.



Figs 14.23A and B: Method of examination of trachea to find out deviation using three fingers.

Horner's syndrome—enophthalmos due to Muller's muscle weakness; drooping of upper eyelid (ptosis); anhidrosis; miosis due to paralysis of dilator pupillae; absence of ciliospinal reflex; flushing of face and nasal congestion. **Causes for Horner's syndrome**—It is due to interruption of sympathetic nerve supply to head and neck. Preganglionic fibres arise from 1st and 2nd thoracic segments of the spinal cord which synapses with three cervical sympathetic ganglia. Any disruption of preganglionic fibres or cervical ganglia or their fibres will cause Horner's syndrome. Causes are—Posterior inferior cerebellar artery thrombosis; often cervical sympathectomy; Pancoast's tumour; secon-



Fig. 14.24: Palpation of carotid artery (common carotid) at the level of thyroid cartilage on the medial border of sternomastoid muscle over Chaisagne tubercle in transverse process of C_6 vertebra. Berry's sign is said to be positive when the pulsation is absent. It signifies advanced carcinoma of thyroid.

daries in the neck; carotid artery aneurysm; spinal cord lesions; injuries to lower root of brachial plexus.

Examination of neck lymph nodes for secondaries should be done. It is commonly palpable in papillary carcinoma of thyroid. It is usually level III and IV nodes. It could be firm, hard or cystic. It is usually brownish black in colour often with papillary projections. Lymph nodes often can get enlarged in follicular carcinoma thyroid and lymphoma. **Lateral aberrant thyroid** which was earlier thought as aberrant thyroid in lateral part of the neck is actually not so but it is secondary in lymph node with primary being papillary carcinoma of thyroid.

Measurement of circumference of the neck at regular intervals is important if patient is not undergoing surgery to assess the increase in size of the thyroid (progress of the swelling) (**Fig. 14.25**).

Percussion over the manubrium sterni is important. Dullness signifies retrosternal extension. Tenderness may signify secondaries in sternum from follicular carcinoma of thyroid. Direct percussion method is the

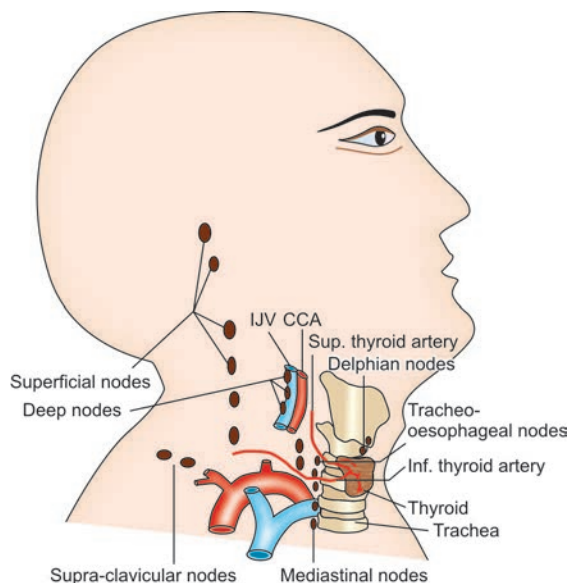


Fig. 14.25: Lymph node drainage of thyroid. Primary and secondary nodes are drainage groups.

usual practice. But it is often painful. Indirect method can also be used (**Fig. 14.26**).

Auscultation over the upper pole of the gland is done to hear bruit—in patients with toxic thyroid and in very vascular tumours (**Figs 14.27A and B**).

Cardiovascular system examination is important in thyrotoxicosis—commonly secondary type. Tachycardia, ectopic beats, pulsus paradoxus, extrasystoles,



Fig. 14.26: Percussion over the sternum is important to rule out retrosternal extension.



Figs 14.27A and B: Bruit over thyroid should be auscultated to find out increased vascularity over upper pole.

atrial fibrillation are the cardiac presentations (**Fig. 14.28**).

Respiratory system examination: Secondaries and pleural effusion can occur in follicular carcinoma of thyroid.

Abdomen examination: Hepatomegaly is looked for as secondaries in liver are known to occur in follicular carcinoma of thyroid. Hepatosplenomegaly can occur as part of Grave's disease or Hashimoto's disease (**Figs 14.29 and 14.30**).



Fig. 14.28: Cardiovascular system is examined and auscultated for cardiac problems in secondary thyrotoxicosis.

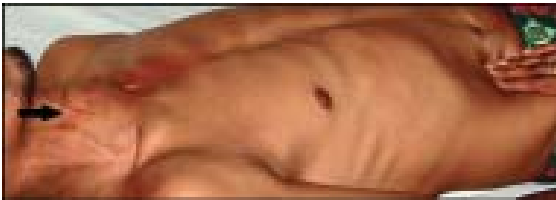


Fig. 14.29: Hepatomegaly can occur in Graves' and Hashimoto's diseases as part of the autoimmune disease.



Fig. 14.30: Palpation of spleen in a patient with thyroid enlargement.

Examination of skull and spine: Localised, warm, vascular, pulsatile secondaries can occur in skull commonly, rib and other bones occasionally as a spread from follicular carcinoma of thyroid (**Fig. 14.31**).

In primary thyrotoxicosis *exophthalmos* and all eye signs are looked for.

Both the eyelids cover the bulbar sclera partially in normal individual. In *lid retraction*—due to spasm



Fig. 14.31: Palpation of skull in thyroid enlargement to look for secondaries when primary is follicular carcinoma of thyroid (pulsatile, vascular, warm, and localised).

of involuntary levator palpebrae superioris muscle, upper eyelid is over or above the upper margin of cornea, often with visible upper bulbar sclera. Here lower eyelid is in normal position. It does not indicate *exophthalmos*. In *exophthalmos lower bulbar sclera is clearly visible* and lower eyelid is below and will not cover the bulbar sclera. In severe *exophthalmos* sclera will be visible all over both above and below (**Figs 14.32A and B**). *Exophthalmos* is measured using exophthalmometer (**Figs 14.33A and B**).

Other Eye Signs

Eye signs are common in primary thyrotoxicosis. Lid lag, lid spasm can occur in secondary thyrotoxicosis also.

1. **Von Graefe's sign:** *Lid lag sign* is visible white sclera above the corneal margin during *lid lag* as upper eyelids cannot keep pace with the eyeball when they look down. Place the examiner's left hand over the patient's head. Place examiner's right index finger near the level of eye and slowly bring it down and ask the patient to see the downward moving finger. If upper sclera is visible then it is positive lid lag sign. Test is repeated few



Figs 14.32A and B: Lid lag check in primary thyrotoxicosis.

more times for confirmation. Normally upper eyelid follows the finger downwards properly but in primary thyrotoxicosis lid lag is observed.

2. **Naffziger's sign:** Examiner stands behind the patient, and patient's neck is extended and examiner looks from behind along the superior orbital



Figs. 14.33A and B: Movements of the eyeball should be checked in primary thyrotoxicosis with exophthalmos to rule out involvement of eyeball muscle due to infiltration by macrophages, inflammatory cells.

margin of the patient. Eyeball is seen beyond the superior orbital margin in exophthalmos (**Fig. 14.34**).

3. **Dalrymple's sign:** Upper eye lid retraction, so visibility of upper sclera.
4. **Stellwag's sign:** Absence of normal blinking — so *staring look*. It is *first sign* to appear. It is due to contraction of striated part of the levator palpebrae superioris in toxic thyroid.



Fig. 14.34: Naffziger's sign.

5. **Joffroy's sign:** Absence of wrinkling on forehead when patient looks up (frowns) with the neck flexed as protruded eyeball obviates the necessity for frowning.
6. **Moebius sign:** Lack of convergence of eyeball. Defective convergence is due to lymphocytic infiltration of inferior oblique and inferior rectus muscles in case of primary thyrotoxicosis. There will be diplopia. It may be an early sign of eventual ophthalmoplegia. Examiner's left hand is placed over the patient's head. Right index finger from distance is brought towards root of the nose between the eyes and patient is asked to follow the approaching finger visually to look for convergence. If positive patient will be unable to converge and develops diplopia (**Fig. 14.35**).
7. **Jellinek's sign:** Increased pigmentation of eyelid margins.
8. **Enroth sign:** Oedema of eyelids (lower eyelid specifically) and conjunctiva.
9. **Rosenbach's sign:** Tremor of closed eyelids.
10. **Gifford's sign:** Difficulty in everting upper eyelid. Differentiates from exophthalmos of other causes.
11. **Loewi's sign:** Dilatation of pupil with weak adrenaline solution.
12. **Knie's sign:** Unequal pupillary dilatation.
13. **Cowen's sign:** Jerky pupillary contraction to consensual light.



Fig. 14.35: Moebius sign.

14. **Kocher's sign:** When clinician places his hands on patient eyes and lifts it higher, patients upper lid springs up more quickly than eyebrows.
15. **Grove's sign.** Upper lid resistance to downward traction.
16. **Rochin's sign:** Reduced amplitude of blinking.
17. **Boston's sign:** Uneven jerky movement of the upper eyelid in inferior movement.
18. **Mean's sign:** Eye globe lags behind upper eyelid on upward gaze.
19. **Griffith's sign:** Lower eyelid lags behind the eye globe on upward gaze.
20. **Sainton's sign:** Frontalis contraction after cessation of levator activity.
21. **Vigourox's sign:** Puffiness of lids.
22. **Ballet's sign:** Ophthalmoplegia—paralysis of more extraocular muscles.
23. **Suker's sign:** Difficulty in maintaining fixation in extreme lateral gaze.
24. **Wilder sign:** Jerking of eyes on movement from abduction to adduction.
25. **Trousseau's/Payne's sign:** Dislocation of the eye globe.
26. **Reisman's sign:** Bruit over eyelid.
27. **Snellen/Donder's sign:** Bruit over the eye.
28. **Goldzieher's sign:** Deep infection of conjunctiva.

Exophthalmos

It is proptosis of the eye, caused by infiltration of the retrobulbar tissues with fluid and round cells, with visible lower bulbar sclera and with *lid spasm* of upper eyelid (Lid spasm is spasm of levator palpebrae superioris muscle which is partly innervated by

Lid retraction is higher upper eyelid with normal lower eyelid with visible sclera adjacent to upper eyelid.

Lid lag is inability of the upper eyelid to keep pace with the eyeball when it looks downwards to follow the examiner's finger.

Exophthalmos is visible sclera first below (lower part) the lower edge of the iris and later eventually upper part of sclera will be visible. It is due to pushing of eyeball forwards due to fat, oedema fluid, cells like macrophages in retrobulbar space.

Order of appearance of signs

1. Stellwag's sign - Mild. First sign to appear
2. Von Graefe's sign - Mild
3. Joffroy's sign - Moderate
4. Moebius sign - Severe

Important signs to be remembered

Visible lower sclera—sign of exophthalmos

Naffziger's sign

VonGraefe's sign—upper lid lag- contraction / overactivity of the involuntary part of the levator palpebrae superioris muscle – Muller's muscle

Joffroy's sign

Moebius sign—most important—early sign of ophthalmoplegia

Thyroid ophthalmopathy in Grave's disease-Werner's abridged classification of ocular changes with van Dyke's modification

Class- grade	Definition
0	No signs and symptoms
1	Eye signs only—refer table below for eye signs
2	Soft tissue involvement
3	Proptosis more than 22 mm
4	Extraocular muscle involvement
5	Corneal involvement- ulceration
6	Loss of sight / vision due to optic nerve and corneal involvement

Eye signs only

Resistance to retro displacement of eye
Oedema of conjunctiva and caruncle
Lacrimal gland enlargement
Infection of conjunctiva
Oedema and fullness of lids

sympathetic fibres). Sclera can be seen clearly below and often above the limbus of the eye. Proptosis can be measured by *exophthalmometer*. Exophthalmos is often self limiting, but not always. Sleeping in propped up position and lateral tarsorrhaphy will help to protect

the eye. *Severe exophthalmos* shows—eyelid oedema, chemosis, conjunctival infection, diplopia, ophthalmoplegia (complete weakness of all extraocular muscles and so no movements possible), corneal ulceration; papilloedema soon develops; finally it may also cause loss of vision. It is called as **malignant exophthalmos** (even though it is neither malignant nor related to any malignancy) (Figs 14.36 to 14.38).

Remember—Antithyroid drugs may worsen exophthalmos and so observe the patient once antithyroid drugs are started as steroids are required to be supplemented.

Grading of exophthalmos



Fig. 14.36: Malignant exophthalmos. Left side tarsorrhaphy is done to prevent corneal ulcer formation.

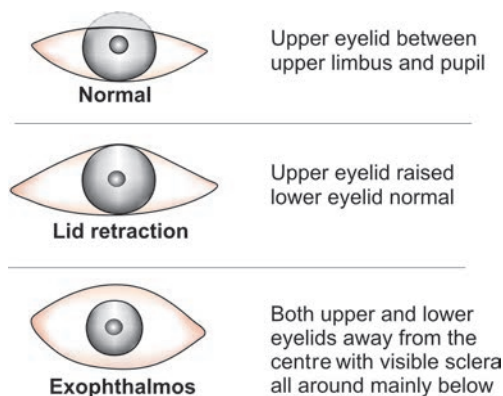


Fig. 14.37: Eyes in different conditions including thyrotoxicosis.

Mild: Widening of palpebral fissure due to lid retraction
Moderate: Orbital deposition of fat causing bulging with positive Joffroy's sign

Severe: Congestion with intraorbital oedema, raised intra-ocular pressure, diplopia and ophthalmoplegia

Progressive: In spite of proper treatment progression of eye signs occurs with chemosis, corneal ulceration and ophthalmoplegia.

<i>Causes of exophthalmos</i>	<i>Causes of pulsating exophthalmos</i>
Endocrinal Thyrotoxicosis—common Cushing's syndrome, acromegaly—rare Congenital deformities of skull Craniostenosis, oxycephaly, hypertelorism Primary tumours Periorbital meningioma Optic nerve glioma Orbital haemangioma Lymphoma Osteoma Pseudotumour—granuloma Secondary tumours Antral carcinoma, neuroblastoma Inflammatory Orbital cellulitis, frontal sinusitis. Vascular causes Cavernous sinus thrombosis/A-V fistula Ophthalmic artery aneurysm Other eye causes Severe myopia Severe glaucoma—buphthalmos	Carotid-cavernous sinus A-V fistula Cavernous sinus thrombosis Orbital vascular neoplasm Orbital haemangioma Ophthalmic artery aneurysm.



Fig. 14.38: Unilateral exophthalmos. Causes other than toxic thyroid have to be thought of—aneurysm of ophthalmic artery, AV malformation, orbital tumours, paranasal sinus tumours, tumours of nasopharynx extending above.

Causes of dyspnoea/stridor in thyroid diseases

Carcinoma thyroid infiltrating recurrent laryngeal nerve/trachea
 Large, long standing goitre causing tracheomalacia
 Retrosternal goitre
 Congestive cardiac failure in thyrotoxicosis

Recent rapid increase in thyroid swelling is due to

Malignant transformation in previous MNG
 Haemorrhage into a nodule
 Anaplastic carcinoma of thyroid.

In a case of thyroid disease following things should be made very clear

Functional status—hyperthyroid/euthyroid/hypothyroid
 Compression to trachea/recurrent nerve
 Neck lymph nodal status
 Tracheal deviation
 Carotid infiltration
 Retrosternal extension
 Systemic features like toxicity or malignant spread to different organs like bone/liver/lungs.

Remember

Goitre is enlargement of the thyroid gland
Solitary nodule is single palpable nodule on clinical examination with rest of the gland not palpable
Dominant nodule is single nodule in a palpable enlarged thyroid gland.
Thyroid swelling is confirmed by its *movement with deglutition* due to attachment of enclosed pretracheal fascia to inferior constrictor muscle which is attached to trachea and cricoid cartilage and so moves with deglutition. Berry's ligament is condensed vascularised pretracheal fascia postero-superomedially. It is important as it is close to recurrent laryngeal nerve.
 Any thyroid swelling *can be malignant* unless proved otherwise
 U/S neck, FNAC, T_3 , T_4 , TSH are essential investigations
 CT scan neck is needed in large goitre and fixed or malignant thyroid
 Radioisotope study I^{123} is done only in selected cases like borderline toxicity, ectopic thyroid, retrosternal goitre and during follow up period after thyroidectomy in follicular carcinoma thyroid to look for secondaries.
 Normal thyroid gland is usually not palpable.

Investigations for Thyroid Diseases

Thyroid Function Tests

- **T₃, T₄, TSH, Free T₃, Free T₄.** T₄ is transported in plasma, binding to thyroxin binding globulin and thyroxin binding prealbumin. Free T₄ is very less. Normal T₄ is 55-150 nmol/litre. T₃ is still lesser than T₄. It is 1.2- 3.1 nmol/litre. Normal TSH is 0-5 IU/ml of plasma. In hyperthyroidism, T₃, T₄ are increased; TSH is decreased/undetectable. T₃, T₄ is decreased; TSH is increased in hypothyroidism. T₃ is more reliable than T₄. Free T₃ and Free T₄ are much more reliable indicators. If one investigation is to be asked, it is better to do TSH estimation. TSH increase is also seen in papillary carcinoma of thyroid. Free T₃ is 0.3% (3-9 pmol/litre). Free T₄ is 0.03% (8-26 pmol/litre).
- **T₃ resin uptake study:** It is the most common indirect method available for the measurement of the proportion of T₄ which is unbound. Patient's serum added with radio labeled T₃ is incubated with ion exchange resin/thyropac. It competes for unoccupied free protein binding sites of T₄. In hyperthyroidism unoccupied free binding sites are low and so resin uptake is low; so resin uptake ratio is less than 85% or low. In hypothyroidism free binding sites are more; so resin uptake is high > 120%. Normal range of resin uptake is 0.9-1.2 µg. 100 × total serum T₄ divided by T₃ resin uptake percentage is called as **free T₄ index**. Normal range is 55-145. Free T₄ index is commonly used; it is single best test available. It is helpful in diagnosing T₃ thyrotoxicosis. Free T₃ index also can be calculated. It is 1.4-3.5.
- **TRH stimulation test** for hypothalamic-pituitary axis: Intravenous TRH (200 µg) shows rise in serum TSH level in 20 minutes (from basal 1 µ unit/ml to 10 µ unit/ml) and reaches to normal in 2 hours. Patients with pituitary insufficiency patients develop a subnormal response; patient with hypothyroidism will show enhanced TSH response; in hyperthyroidism there will be no response. This test is useful in doubtful hyperthyroidism, hypothyroidism, T₃ thyrotoxicosis, ophthalmic Graves' disease. Drugs like L thyroxine, steroids, oestrogens, levodopa interfere with the response.
- **Protein bound iodide (PBI):** It is cheaper but nonspecific and unreliable test as it also measures nonhormonal forms of iodide. Normal value is 4 - 8 µg/100 ml. False positivity is seen in pregnancy, use of iodide containing cough syrups, iodide containing X-ray contrast, and oral contraceptives. False negativity is found with use of salicylates, androgens, hydantoins.
- **Radioisotope studies:**
 - Uptake study** - Thyroid traps iodine and rate reflects hormone secretion. Uptake is measured in 10-120 minutes. Later protein bound I¹³¹ is measured. Dose of I¹³¹ is 5µ curie. Half life of I¹³¹ is 8 days; I¹³² is 2.3 hours; I¹²³ is 13 hours. Now Technetium ⁹⁹ is also used as it has short half life with very less radiation dose. It gets concentrated similar to iodine isotopes and does not bind to tyrosine and so precise iodine trap can be assessed. Uptake study cannot be done after contrast X-rays like IVU. It takes 2 weeks to excrete contrast after IVU; 4 weeks after cholecystogram; many years after bronchography and myelography.
 - Thyroid scan**—It is done using I¹²³/I¹³¹ or ^{99m}Tc. It is not done in every patient with thyroid disease. It is indicated in solitary nodule; borderline toxicity; retrosternal goitre; ectopic thyroid; to study the metastases in entire body in functioning thyroid carcinomas after total thyroidectomy. ^{99m}Tc is injected intravenously. I¹²³/I¹³¹ is given orally. When ^{99m}Tc is used scan is done in half an hour. When iodine radioisotopes are used, scan is done in 24 hours. Cold nodule is non-functioning nodule; hot nodule is hyperfunctioning nodule; warm nodule is normal functioning gland. In autonomous nodule, nodule is hot and rest of gland will not show any activity. In functioning nodule which is not autonomous, nodule as well as remaining gland will show the activity.
- **Werner's T₃ suppression test:** Initial isotope uptake study is done. 40 µg of T₃ is given to the patient orally 8th hourly for 5 days. Uptake study is repeated. In normal uptake suppression upto 80% is noted. In toxic goitre suppression is 10 - 20%. It is used in patients with antithyroid drugs for primary thyrotoxicosis to assess the remission status.
- **Other tests:** BMR (increased in toxicity); Serum cholesterol (decreases in toxicity); serum creatinine (increases in toxicity).

U/S neck for thyroid and neck nodes. X-ray neck to see calcifications (*speckled fine* calcification in papillary carcinoma of thyroid; *coarse, ring-like* calcification in MNG) and tracheal deviation.

FNAC thyroid and lymph node.

CT neck in malignancies or large goitre.

Trucut biopsy if two trials of FNAC are inconclusive. It can injure deeper structures like recurrent laryngeal nerve and also can cause hemorrhage.

Frozen section biopsy on table and proceed may be needed to rule out malignancies.

Special blood analysis: Serum calcitonin, serum thyroglobulin estimation in neoplasms of thyroid.

Role of ultrasound (U/S) in thyroid diseases

To detect number, size, nature of the nodules (cystic/solid/complex) (complex means cystic and solid together—more suspicious of carcinoma). Size up to 2 mm can be detected.

U/S guided FNAC is very useful.

U/S at regular intervals is advisable to observe a small nodule in thyroid.

To detect recurrent nodule.

To find out the invasion/spread/vascularity/status of carotid artery and internal jugular vein.

To find out enlarged lymph nodes in neck.

Role of FNAC in thyroid swelling

Highly sensitive in papillary carcinoma of thyroid and also its nodal spread.

Useful to differentiate between benign and malignancy (**Fig. 14.39**).

Useful in lymphoma/anaplastic carcinoma/medullary carcinoma thyroid/Hashimotos thyroiditis.

It is not very useful in follicular carcinoma as it is difficult to differentiate it from follicular adenoma as capsular invasion/vascular invasion is main feature in follicular carcinoma.

Note:

On table frozen section biopsy is useful in negative FNAC but doubtful cases. Definitive procedure is undertaken once frozen section report comes on table. But in frozen section biopsy itself, 15% of follicular carcinoma report may be inconclusive or negative which causes difficulty in taking decision. In such occasion hemithyroidectomy is done and once histology report of follicular carcinoma is obtained completion thyroidectomy is done usually immediately within a week. If biopsy report is delayed then completion thyroidectomy is done after 6 weeks.



Fig. 14.39: FNAC thyroid is an important investigation in thyroid diseases.

Trucut biopsy gives tissue diagnosis but danger of haemorrhage and injury to vital structures like trachea, recurrent laryngeal nerve, vessels are likely.

Solitary Thyroid Nodule

It is a *single palpable nodule* in thyroid on *clinical examination*, in an otherwise normal gland. **Causes:** (1) Thyroid adenomas—Follicular—common (40% of *actual single nodule* excluding solitary nodule of MNG); Hurthle cell. (2) Papillary carcinoma of thyroid—15%. (3) Only one nodule palpable in an underlying multinodular goiter – *commonest* cause *only clinically*—50%. (4) Thyroid cyst.

Types:

Based on function: (1) Toxic solitary nodule. (2) Non-toxic solitary nodule.

Based on radioisotope study: (1) *Hot*—Means autonomous toxic nodule. (2) *Warm*—Normally functioning nodule. (3) *Cold*—Non-functioning nodule; may be malignant-20% (need not be always). Cold nodule may be due to malignancy, thyroiditis, thyroid cyst or haemorrhage. (4) *Hot or warm in ^{99m}Technetium scan but cold in I¹²³ scan*—commonly they are malignant.

Note: Thyroid nodule in children and elderly can be malignant. Rapid enlargement of thyroid nodule can be malignant. 30% of solitary nodules are cystic.

Features: Single nodule palpable in one or other lobes of the thyroid which is usually smooth, globular, firm

with well-defined margin. Overlying skin is normal. *Lahey's test* does not show any other nodules in posterior part of the gland. Tracheal deviation towards opposite side is common—confirmed by trail sign, three finger test and auscultation. U/S neck is very useful. FNAC is essential. When FNAC is inconclusive Trucut needle biopsy may be done but it can cause pain/bleeding/recurrent laryngeal nerve injury. T_3 , T_4 , TSH are done to find out the function. Radioisotope study (^{123}I / ^{131}I / $^{99\text{m}}\text{Tc}$) may be often needed. CT scan or MRI neck is done only in selected cases but not routinely (large swelling/to see vascularity/retrosternal extension are the indications) (Figs 14.40A to 14.42).

Indications for surgery in solitary nodule thyroid

Malignant nodule
Follicular neoplasm
Toxic nodule in young
Nodules with obstruction
Recurrent cystic nodule
Complex cyst (both solid and cystic component)
Cosmetics.

Possible features of suspected malignancy in solitary nodule thyroid

Any nodule can be malignant whether nodule is hard/firm/cystic/small/large/asymptomatic
Rapid onset/rapid recent increase in size
Hoarseness of voice/dysphagia/stridor/dysphagia
Fixity of the nodule
Palpable significant neck nodes.

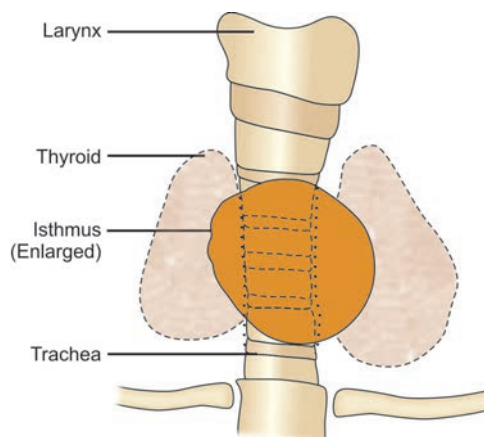
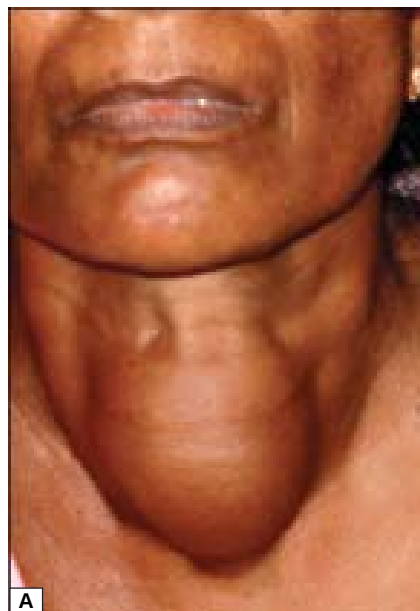
Diffuse Hyperplastic Goitre

Initial persistent increase in TSH level causes diffuse active lobules. In late stages of diffuse hyperplasia, TSH stimulation decreases and many follicles become inactive and gets filled with colloid, called as *colloid goitre*. As *diffuse hyperplastic goitre* is a reversible stage, *l-thyroxine* is beneficial.

Nodular Goitre

Pathogenesis

Persistent TSH stimulation → Diffuse hyperplasia of gland (all active lobules) → Later with fluctuation of TSH level → Mixed areas of active and inactive lobules develop → Active lobules become more vascular and hyperplastic → Haemorrhages occur with necrosis in the centre → Nodule formation → Centre of nodule is inactive and only margin is active, i.e.

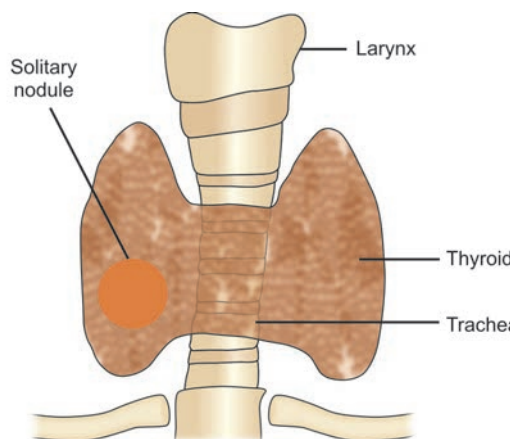


Figs 14.40A and B: Solitary nodule involving isthmus.

internodular tissue is active → Formation of many nodules → *Multi-Nodular Goitre (MNG)*. Other factors involved are growth stimulating immunoglobulins and growth prone cell clones.

Features

It is a slowly progressive disease with many years of history; multiple nodules of different sizes are formed in both lobes, also in isthmus, which is firm,



A

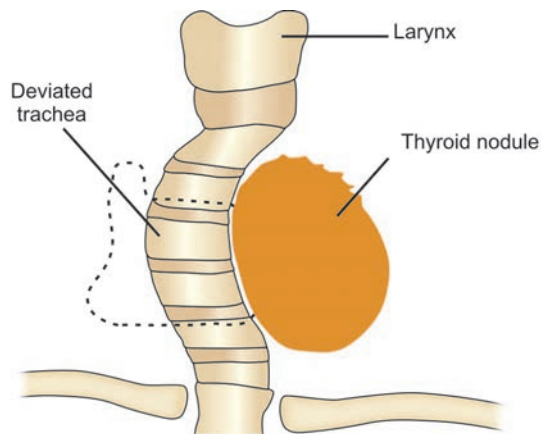


Fig. 14.42: Solitary nodule thyroid causes deviation of trachea to opposite side.



Figs 14.41A and B: Solitary nodule right lateral lobe. It is a clinical entity.

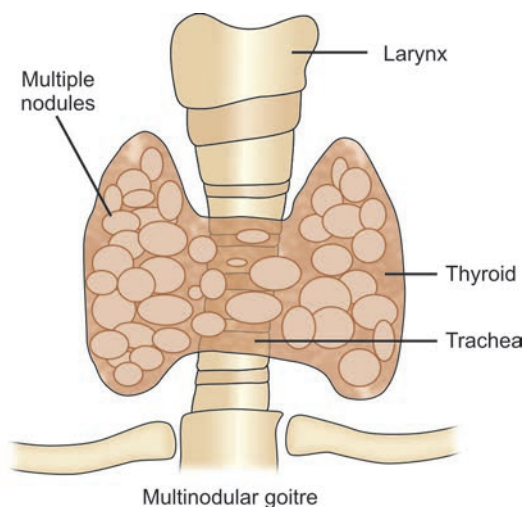


Fig. 14.43: Diagrammatic representation of multinodular goitre.

nodular, nontender, moves with deglutition; recent increase in size signifies malignant transformation or haemorrhage (**Figs 14.43 and 14.44**). Investigations are T_3 , T_4 , TSH, U/S neck, FNAC, X-ray neck shows ring or rim calcification.

Complications of MNG

Secondary thyrotoxicosis (30%); follicular carcinoma of thyroid (10%); haemorrhage in a nodule; tracheal obstruction; calcification.

Thyroid cyst

It is thyroid swelling which is cystic in nature eliciting positive fluctuation.

Common cause is colloid degeneration.

30% of solitary nodules are cystic.

15% cystic swellings in thyroid are malignant.

A cyst if contains both solid and cystic areas is called as *complex cyst* which is more likely to be malignant. FNAC may cause regression in simple cyst. Surgery is needed if recurrence occurs after three repeated aspirations.

Surgery is indicated in complex cyst and if cyst is more than 4 cm in size.



Fig. 14.44: Large multinodular goitre.

Thyrotoxicosis and Hyperthyroidism

It is complex of symptoms and signs due to raised levels of thyroid hormones.

Types

(1) Diffuse toxic goitre-(Grave's disease, Basedow's disease, Primary thyrotoxicosis). (2) Toxic multinodular goitre (Secondary thyrotoxicosis; *Plummer disease*). (3) Toxic nodule. (4) Hyperthyroidism of rarer causes: *Thyrotoxicosis factitia*-drug induced due to intake of L-thyroxine more than normal; *Jod Basedow thyrotoxicosis*—because of consumption of large doses of iodides given to a hyperplastic endemic goitre; *Autoimmune thyroiditis* or *de Quervain's thyroiditis*; Occasionally *carcinoma thyroid*; *Neonatal thyrotoxicosis*—It subsides in 3-4 weeks as TsAb titres fall in the baby's serum. **Note: Wolf-Chaikoff effect**—iodides inhibit the further release of hormone causing hypothyroidism. It is eight times more common in females; occurs in any age group; primary type is seen commonly in younger age group; secondary is common in older age group. **Grave's disease** is an autoimmune disease with increased levels of specific antibodies in the blood (TSH receptor antibodies). It is often associated with vitiligo. It is often familial. Thyroid stimulating immunoglobulins (TSI)/thyroid

stimulating antibodies (TsAb) and long acting thyroid stimulator (LATS) cause pathological changes in the thyroid. Histologically there is acinar cell hypertrophy and hyperplasia with absence of normal colloid in the tall columnar epithelium (normal is flat epithelium with colloid). As cells are empty, they look vacuolated. Tissues are highly vascular. Exophthalmos producing substance (EPS) causes Grave's ophthalmopathy (**Fig. 14.45**).



Fig. 14.45: Thyrotoxicosis (primary). Note the exophthalmos and also toxic thyroid enlargement.

Symptoms of Hyperthyroidism

Gastrointestinal system: Weight loss in spite of increased appetite; diarrhoea (due to increased activity at ganglionic level).

Cardiovascular system: Palpitations, chest pain; shortness of breath at rest or on minimal exertion; angina; irregularity in heart rate; cardiac failure.

Neuromuscular system: Undue fatigue and muscle weakness, exaggerated tendon reflexes, myasthenia like syndrome; tremor, hyperkinesias, increased sweating.

Skeletal system: Increase in linear growth in children.

Genitourinary system: Oligo- or amenorrhoea; occasional urinary frequency.

Integument: Hair loss; pruritus; palmar erythema.

Wayne's Diagnostic Indices (Clinical)			
Symptoms		Present	Absent
1	Dyspnoea on effort	+1	−5
2	Palpitation	+2	
3	Tiredness	+2	
4	Preference for heat		
5	Preference for cold (Heat intolerance)	+5	
6	Excessive sweating	+3	
7	Nervousness	+2	
8	Appetite increased	+3	
9	Weight decreased	+3	
Signs		Present	Absent
1	Bruit over thyroid	+2	−2
2	Exophthalmos	+2	
3	Lid retraction	+2	
4	Lid lag	+1	
5	Hyperkinetic movements	+4	
6	Fine finger tremors	+1	
7	Hands Hot	+2	
	Moist	+1	
8	Atrial fibrillation	+4	
9	Pulse rate 80/minute.		
	80-90/minute	0	
	More than 90/minute	+3	
10	Palpable thyroid	+3	
< 11 points –non toxic		11-19 –equivocal	

Psychiatry: Irritability; nervousness; insomnia. *Sympathetic overactivity* causes dyspnoea, palpitation, tiredness, heat intolerance, sweating, nervousness, increased appetite and decrease in weight. Because of the *increased catabolism* they have increased appetite, decreased weight and so also increased creatinine level which signifies *myopathy* (due to more muscle catabolism). *Fine tremor* is due to *diffuse irritability of grey matter*. Thrill is felt in the upper pole of the thyroid and also bruit is heard on auscultation. It is because superior thyroid artery enters the gland superficially in upper pole, and so thrill and bruit can easily be assessed. Inferior thyroid artery enters the gland in lower pole at deeper plane and so thrill cannot be felt (**Fig. 14.46**).

Signs of Hyperthyroidism

(1) *Eye signs* in toxic goitre (Refer above). (2) *Cardiac manifestations*: Tachycardia is common. Sleeping pulse rate is usually checked for three consecutive

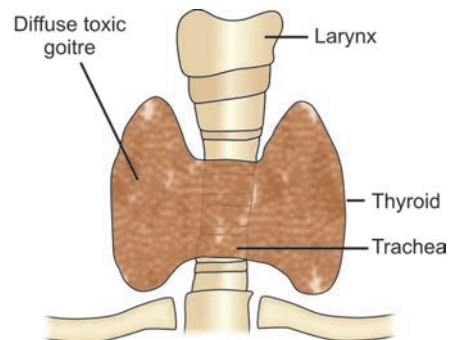


Fig. 14.46: Diffuse toxic goitre. Note the involvement of both lobes bilaterally.

nights and average is taken as the value; ectopic; pulsus paradoxus; wide pulse pressure; multiple extrasystoles; paroxysmal atrial tachycardia; paroxysmal atrial fibrillation; persistent atrial fibrillation (not responsive to digoxin). (3) *Myopathy*: Weakness of proximal muscles occurs, i.e. the front thigh muscles, or arm muscles; weakness is more when

muscle contracts isometrically, i.e. either while climbing down steps, or lifting a full bucket etc. often when it is severe it resembles myasthenia gravis. Once hyperthyroidism is controlled recovery occurs. (4) *Pretibial myxoedema* is often a feature of primary thyrotoxicosis (It is a misnomer)—It is usually symmetrical, shiny, red thickened skin, with coarse hair; in severe cases skin of whole leg below the knee with foot and ankle is involved: it is due to deposition of myxomatous tissues (mucin like deposits) in skin and subcutaneous plane; it might or might not regress completely after treatment for toxicity; it is associated with exophthalmos with high levels of thyroid stimulating antibodies. (5) *Thyroid Acropachy* is clubbing of fingers and toes in primary thyrotoxicosis; hypertrophic pulmonary osteoarthropathy also may develop.

Cardinal signs of toxic thyroid

- Palpable thyroid often with thrill and bruit
- Tremor of hands and tongue
- Tachycardia
- Exophthalmos

Toxic nodule: It is a solitary overactive nodule. There is an autonomous hypertrophy and hyperplasia of the part of the gland where there is a nodule. [It is not due to Thyroid stimulating antibody (Ts Ab)]. Here high levels of circulating thyroid hormones suppress TSH secretion and so normal thyroid tissue surrounding the nodule is been suppressed and inactive.

Toxic thyroid in pregnancy and children: Radioiodine therapy is absolutely contraindicated in pregnancy (High risk to foetus) and children (high risk of developing thyroid carcinoma).

Thyrocardiac: Severe cardiac damage resulting from hyperthyroidism usually secondary type requires proper opinion from cardiologists and treatment with propranolol.

T₃ thyrotoxicosis should be suspected if the clinical picture is suggestive of toxicosis, but routine tests for thyroid function are within normal range.

Investigations

(1) Serum T₃ and T₄ levels are very high. TSH is very low or undetectable. Sometimes, only T₃ level is increased and is called as T₃ toxicosis. In T₃ toxicosis, *free T₃ estimation is important*. Radioisotope study by I¹³¹ will show more uptake, i.e. *hot nodules* or hot areas. This is very useful to detect autonomous solitary toxic nodule. (2) TRH estimation. (3) ECG—To look for cardiac involvement and if required opinion from cardiologists' is taken and cardiac problems are managed accordingly. (4) Total count and neutrophil count are very essential base line investigations before starting antithyroid drugs (as it may cause agranulocytosis).

Differential Diagnosis of Thyrotoxicosis

Anxiety neurosis—hands are cold and moist, sleeping pulse rate is normal, thyroid enlargement is not present; phaeochromocytoma; malabsorption syndrome; diabetes mellitus.

Thyroid Neoplasms

Classification of Thyroid Neoplasm

Benign: Follicular adenoma; Hurthle cell adenoma; colloid adenoma—commonest; papillary adenoma—its existence is doubtful. It is invariably low grade

Differentiating Points Between Primary and Secondary Hyperthyroidism

Primary thyrotoxicosis	Secondary thyrotoxicosis
1. Symptoms appear first, then swelling. 2. Goitre is diffuse, smooth, firm or soft, both lobes are involved 3. There is thrill and bruit 4. Features are much more severe compared to that of secondary toxicosis. 5. Eye signs and exophthalmos are common. 6. As it is an autoimmune disease, there may be hepatosplenomegaly. <i>Histologically, there is hyperplasia of acini, lined by columnar epithelium, often containing vacuolated colloid.</i>	1. Swelling appears first 2. Swelling is large nodular, obvious 3. Symptoms appear after long time, which is less severe and slowly progressive compared to primary toxicosis. 4. Cardiac features are more common. 5. Eye signs are not common.

papillary carcinoma.

Malignant (Dunhill classification): (a) *Differentiated*—Papillary carcinoma (60%); Follicular carcinoma (7%); papillofollicular carcinoma behaves like papillary carcinoma of thyroid; Hurthle cell carcinoma behaves like follicular carcinoma. (b) *Undifferentiated*. Anaplastic carcinoma (13%). (c) *Medullary carcinoma* (6%). (d) *Malignant lymphoma* (4%). (e) *Secondaries* in thyroid (rare)—from colon, kidney, melanoma (**Fig. 14.47**).

Aetiology of thyroid malignancy: (1) Radiation either



Fig. 14.47: Carcinoma thyroid. Note the superficial tissue infiltration. Dilated veins are also seen.

external or radioiodine can cause papillary carcinoma thyroid. There was increased incidence of thyroid carcinoma among children following exposure to ionizing radiation after the Chernobyl nuclear disaster in Ukraine in 1986. Irradiation to head and neck region used to be the therapy for benign conditions like adenoids, acne vulgaris, thymus enlargement, haemangiomas which predisposed papillary carcinoma of thyroid. Radiotherapy for Hodgkin's lymphoma in younger age group may later cause papillary carcinoma of thyroid. (2) Pre-existing multinodular goitre. It turns into follicular carcinoma of thyroid. (3) Medullary carcinoma of thyroid commonly and 6% of papillary carcinoma of thyroid can be familial. (4) Hashimoto's thyroiditis may predispose to papillary carcinoma of



Fig. 14.48: Carcinoma thyroid in a male patient showing vascularity.

thyroid and also NHL (**Fig. 14.48**).

Papillary Carcinoma

It is 60% common; common in females (3: 1) and young age group. TSH levels in the blood of these patients are high and so it is called as *hormone* dependent tumour. It is a slowly progressive and less aggressive tumour. It is commonly multicentric. It spreads within the gland through intrathyroidal lymphatics to other lobe, comes out of the capsule and spreads to lymph nodes. Usually there is no blood spread.

Types: Occult (< 1.5 cm); Intrathyroidal; Extrathyroidal; Micropapillary carcinoma is less than 1 cm in size or clinically not detectable.

Gross: It can be soft, firm, hard, and cystic. It can be solitary or multinodular. It contains brownish black fluid.

Microscopy: It shows cystic spaces, papillary projections with *psammoma* bodies, malignant cells with 'Orphan Annie eye' nuclei (intranuclear cytoplasmic inclusions, nuclear grooving).

Clinical features: (1) Soft or hard or firm, solid or cystic, solitary or multinodular thyroid swelling. (2) Compression features are uncommon in papillary carcinoma thyroid. (3) Often discrete lymph nodes in the neck are palpable.

Diagnosis: FNAC of thyroid nodule and lymph node,

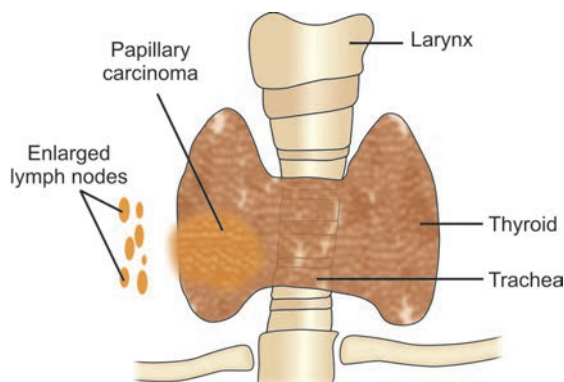


Fig. 14.49: Papillary carcinoma of thyroid with lymph nodal spread.

to see psammoma bodies, nuclear changes; Radio-isotope scan shows cold nodule; TSH level in the blood is higher (**Fig. 14.49**).

AMES scoring

- A: Age less than 40 years has got better prognosis.
- M: Distant metastasis.
- E: Extent of the primary tumour.
- S: Size of the tumour. Size less than 4 cm has got better prognosis.

AGES scoring

- A: Age less than 40 years has got better prognosis.
- G: Pathologic grade of the tumour.
- E: Extent of the primary tumour.
- S: Size of the primary tumour. Size less than 4 cm has got better prognosis.

Psammoma bodies are seen in

- Papillary carcinoma thyroid
- Meningioma
- Serous cystadenoma of ovary

Berry's in thyroid

- Berry ligament
- Berry sign
- Berry picking

Lateral aberrant thyroid is a misnomer. It is actually secondaries in neck lymph nodes which are palpable from an occult primary from papillary carcinoma of thyroid (which is clinically not palpable).

Thyroid Paradox

Cellular tumours are soft, and cystic tumours are firm or hard (tensely cystic). It is observed in papillary carcinoma of thyroid.

Features of thyroid carcinoma

Any thyroid of any size, any texture—solid/cystic, with nodules of any number—single/multiple, in any age group can be malignant

Features of infiltration

Infiltration of strap muscles often with sternomastoid muscle
 Infiltration of laryngotracheal complex causing stridor and often haemoptysis
 Infiltration of recurrent laryngeal nerve causes hoarseness of voice
 Infiltration of oesophagus causes dysphagia/odynophagia (painful swallowing)
 Infiltration into carotid sheath causes absence of carotid pulsation—*Berry's sign*
 Infiltration of cervical sympathetic chain causing Horner's syndrome
 Rarely infiltration into cranial nerves or brachial plexus can occur

Features of lymph nodal spread

Discrete neck node involvement can occur commonly in papillary carcinoma of thyroid, often in medullary carcinoma and occasionally in follicular carcinoma. Lymph node is often cystic (20%) and contains brownish-black material in papillary carcinoma.
 Central neck (level VI) and mediastinal nodes often can get involved in thyroid malignancy. Primary nodes may be involved but clinically not palpable. Superior mediastinal nodes (level VII) can cause compression of SVC, recurrent laryngeal nerve with often dullness in the sternum. These nodes can get involved without palpable neck nodes. In the neck, palpable nodes are commonly levels—II, III and IV occasionally level V. Secondary nodes—clinically palpable.
 Only palpable neck node may be presentation without clinically palpable thyroid—secondary of occult primary (papillary) thyroid carcinoma. FNAC of the node concludes the diagnosis.
 Central node dissection is the common practice while doing total thyroidectomy in carcinoma thyroid especially in medullary carcinoma of thyroid.

Features of blood spread

Follicular carcinoma commonly spreads through blood to bone, lungs, and liver. Bone secondary is typical. It is well-localised, smooth, soft/hard, warm, nonmobile, vascular and pulsatile. It is common in the skull bone—frontal/parietal bone. It can occur in other bones also. Lung secondaries present with chest pain, dyspnoea and haemoptysis.
 Liver secondaries cause hepatomegaly and jaundice. Blood spread also can occur in medullary carcinoma of thyroid.

Follicular Carcinoma

It is 17% common. It is common in females. It can occur either *de novo* or in a pre-existing multinodular goitre. It is a more aggressive tumour. It spreads mainly through blood into the lung, bones, liver. Bone secondaries are typically vascular, warm, pulsatile, localised, commonly in *skull*, long bones, ribs. It can also spread to lymph nodes in the neck (10%) occasionally.

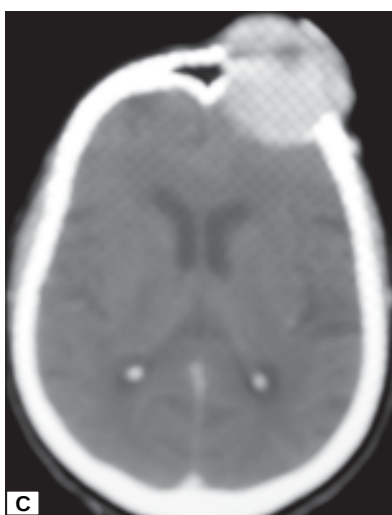
Types: *Non-invasive*—blood spread is not common.
Invasive—blood spread is common.

Typical feature: *Angioinvasion and capsular invasion* (Figs 14.50 and 14.51A to C).

Clinical features: Swelling in the neck, firm or hard and nodular; tracheal compression/infiltration and stridor; dyspnoea, haemoptysis, chest pain when there are lung secondaries; recurrent laryngeal nerve involvement causes hoarseness of voice, positive 'Berry's sign' signifies advanced malignancy (Infiltration into the carotid and so absence of carotid pulsation); pulsatile, warm, well localised, vascular secondaries in the skull (frontal/parietal bones), long bones.



Fig. 14.50: Follicular carcinoma of thyroid in a male patient. It is involving mainly left lobe but it is extensive and spreading to adjacent soft tissues.



Figs 14.51A to C: Follicular carcinoma of thyroid causing secondaries in skull. It is localized, vascular, smooth, pulsatile, warm secondaries (in skull). CT scan is head diagnostic.

Investigations: Most often FNAC is inconclusive, because capsular and angioinvasion which is the main feature in follicular carcinoma cannot be detected by FNAC. Frozen section biopsy is very useful. But in 15% cases it shows negative results. U/S abdomen, Chest X-ray, X-ray bones are the other investigations required (**Fig. 14.52**).



Fig. 14.52: X-ray skull showing secondaries from follicular carcinoma thyroid.

Hurthle cell carcinoma is a variant of follicular carcinoma of thyroid which contains abundant oxyphil cells. It spreads more commonly to regional lymph nodes than follicular carcinoma of thyroid. ^{99m}Tc **sestamibi scan** is very useful for Hurthle cell carcinoma. It does not take up I¹³¹; has got poorer prognosis than follicular carcinoma.

Differential diagnosis for carcinoma thyroid

Multinodular goitre
Solitary nodule of other causes
Riedel's thyroiditis.

Note: Toothpaste colloid—follicular carcinoma; Chewing gum colloid—papillary carcinoma; Nuclear grooving—papillary carcinoma; Psammoma bodies—papillary carcinoma; Amyloid—medullary carcinoma; Follicular adenoma—aneuploid and in follicular carcinoma—diploid—DNA ploid study (**Fig. 14.53**).

Anaplastic Carcinoma

It is a very aggressive tumour of short duration, presents with a swelling in thyroid region which is rapidly progressive causing—stridor and hoarseness of voice; dysphagia; fixity to the skin; infiltration into the carotid sheath—positive Berry's sign; swelling is



Fig. 14.53: Carcinoma thyroid with dilated veins on the surface.

TNM staging for thyroid cancer

Tumour

Tx Primary not assessed
T0 No primary
T1 < 1 cm limited to thyroid
T2 1-4 cm limited to thyroid
T3 > 4 cm limited to thyroid
T4 Any size extending beyond capsule.

Nodes

Nx Nodes cannot be assessed
N0 No regional nodes
N1 Regional nodes present
N1a Same side neck nodes
N1b Bilateral, midline, mediastinal nodes

Metastases

Mx Cannot be assessed
M0 No metastases
M1 Metastases present

Under the age of 45 years only Stage I —[any T/N and M0] and II [any T/N and M1]. After the age 45 years, Stage I, II, III and IV. Anaplastic carcinoma is always stage IV.

hard, with involvement of isthmus and bilateral lobes; FNAC is diagnostic; tracheostomy and isthmectomy has got a role to relieve respiratory obstruction temporarily. It carries poor prognosis (**Figs 14.54 and 14.55**).

Medullary Carcinoma of Thyroid (MCT)

It is uncommon (5%) type of thyroid malignancy. It arises from the *para-follicular 'C' cells* which is

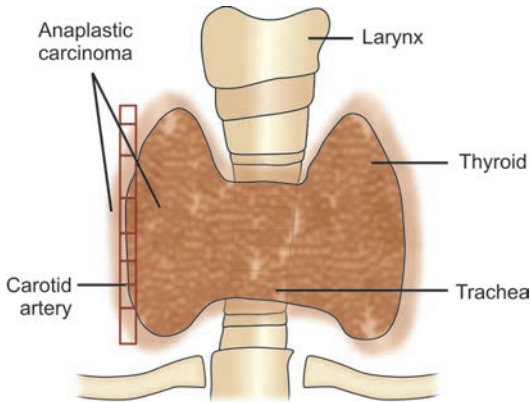


Fig. 14.54: Anaplastic carcinoma of thyroid with infiltration through the capsule. It commonly encases the carotid artery causing positive Berry's sign.



Fig. 14.55: Anaplastic carcinoma of thyroid.

derived from the ultimobronchial body (neural crest). They are part of APUD (Amine Precursor Uptake Decarboxylation) cells. C cells are more in upper pole of the thyroid. It contains characteristic 'amyloid stroma' wherein malignant cells are dispersed. In these patients blood levels of *calcitonin* both basal as well as that following calcium or pentagastrin stimulation is high, a very useful tumour marker. Tumour also secretes 5-H.T (serotonin), prostaglandin and vasoactive intestinal polypeptide (VIP). It spreads mainly to lymph nodes (60% common). It may be associated with MEN II syndrome and pheochromocytoma with hypertension. There may be

mucosal neuromas in lips, oral cavity.

Clinical features: Thyroid swelling often with enlargement of neck lymph node; diarrhoea, flushing; hypertension, pheochromocytoma and mucosal neuromas when associated with MEN II syndrome. Sporadic and familial types occur in adulthood whereas cases associated with MEN syndrome II occur in younger age groups.

Types: (1) *Sporadic*. Usually solitary—70%. (2) *MCT with MEN II syndrome*. MCT with MEN II B with Pheochromocytoma is most aggressive type. (3) *Familial MCT*—It is autosomal dominant with proto-oncogene in chromosome number 10. It is commonly multicentric.

Investigations: FNAC: shows amyloid deposition with dispersed malignant cells and 'C' cell hyperplasia; Tumour marker: Calcitonin level will be higher. Normally it is less than 0.08 ng/L; U/S neck-thyroid region; Urinary VMA, urinary catecholamines, urinary metanephrine, serum calcium, serum parathormone estimation; CT neck and chest to evaluate nodal status is a must; Indium¹¹¹ octreotide scanning is useful in detecting medullary carcinoma thyroid (70% sensitivity). It is also useful in postoperative follow up to find out residual/metastatic disease.

If there is associated pheochromocytoma it should be treated first surgically by adrenalectomy and later total thyroidectomy is done. *All family members of the patient should be evaluated for serum calcitonin and if it is high they should undergo prophylactic total thyroidectomy* (Can also be assessed by genetic evaluation.) If there is positive RET proto-oncogene in MCT with MEN IIA and familial MCT types, prophylactic total thyroidectomy is done at the age of **5 years**. In positive RET proto-oncogene in MCT with MEN IIB prophylactic total thyroidectomy is done at the age of **one year**. MCT when associated with parathyroid hyperplasia (30%) in MEN IIA, total thyroidectomy with central nodal dissection with total parathyroidectomy is done followed by autotransplantation of half of gland in sternomastoid or non-dominant forearm brachioradialis muscle.

Malignant lymphoma: It is NHL type. Occurs in a

pre-existing Hashimoto's thyroiditis (Not proved well). FNAC is useful to diagnose the condition.

Hashimoto's Thyroiditis (Struma Lymphomatosa)

It is also called as diffuse non-goitrous thyroiditis. It is an autoimmune thyroiditis which is common in women. Initially there is hyperplasia, then fibrosis, eventually infiltration with plasma cells and lymphocytic cells. Askanazy cells are typical (like Hurthle cells). The river Struma arises in Bulgaria and flows into Aegean Sea. Struma means goitre. Banks of this river are endemic area for goitre.

Clinical features: Painful, diffuse, enlargement of usually both lobes of thyroid which is firm, tender and smooth; initially they present with toxic features but later, they manifest with features of hypothyroidism. There may be hepatosplenomegaly; it is often associated with other autoimmune diseases; in 85% cases significant rise in the thyroid antibodies (microsomal, thyroglobulin, or colloid antibodies) is observed; papillary carcinoma may develop in Hashimoto's thyroiditis; often condition may be associated with or may predispose to *malignant lymphoma*. At present it is not well proved.

de-Quervain's Subacute Granulomatous Thyroiditis

It is due to viral aetiology either mumps or Coxsackie viruses causing inflammatory response with infiltration of lymphocytes, neutrophils, multinucleated giant cells.

Clinical features: Pain is diffuse, swelling in thyroid which is tender; commonly seen in females; initially there will be transient hyperthyroidism with high T_3 and T_4 but poor radioiodine uptake; *it is usually a self-limiting disease*.

Riedel's Thyroiditis (Woody Thyroiditis; Ligneous Thyroiditis; 0.5% common)

A very rare benign entity wherein thyroid tissue is replaced by fibrous tissue which interestingly infiltrates the capsule, extends into muscles, paratracheal tissues, and carotid sheath. It is often associated with retroperitoneal and mediastinal fibrosis.

Clinical features: Stony hard, fixed, small swelling in a male with stridor, often Berry's sign may be

positive, i.e. absence of carotid pulsation. Movement with deglutition often may be difficult to elicit.

Differential diagnosis: Anaplastic carcinoma of thyroid.

Investigations: T_3 , T_4 may be low due to hypothyroidism; radioisotope scan will not show any uptake; FNAC to rule out carcinoma.

Thyroid Steal

Patient is taken to operation theatre for few days before doing surgery so as to reduce the anxiety of the patient. Eventually steal the patient for surgery.

Note: Condition resulting from total removal of thyroid was called as *Cachexia strumipriva* by Kocher.

Thyrotoxic Crisis (Thyroid Storm)

It occurs in a thyrotoxic patient inadequately prepared for thyroidectomy and rarely a thyrotoxic patient presents in a crisis following an unrelated operation or stress. They present in 12-24 hours with severe dehydration due to circulatory collapse, hypotension, hyperpyrexia and often cardiac failure. *Treatment* is injection hydrocortisone, oral antithyroid drugs, tepid sponging of whole body, beta blocker injection, oral iodides, large amount of IV fluids for rehydration, digitoxin, cardiac monitor, often ventilator support and observation. It has got high mortality rate with critical period of 72 hours. Fluid and electrolyte management, cardiac management are important aspects to be monitored and treated.

Thyroglossal Cyst

Thyroglossal cyst is a swelling occurring in the neck in any part along the line of thyroglossal tract. It is a tubulodermoid. It is accumulation of the cystic fluid secreted by the portion of the unobliterated part of the thyroglossal duct/tract.

Possible Sites for Thyroglossal Cyst

Beneath the foramen caecum; in the floor of mouth; suprahyoid; *subhyoid—commonest site*; on the thyroid cartilage; at cricoid cartilage level—here tug feel may not be elicited and so difficult to differentiate from adenoma of thyroid isthmus. It is usually congenital wherein there will be degeneration of a part of the

tract causing cystic swelling. Normal thyroid may be present in the normal location (fossa). Sometimes, thyroid may not be present in the normal site but may be present in the wall of the thyroglossal cyst. It contains gel-like fluid. It is lined by columnar epithelium surrounded by lymphoid tissues.

Clinical Features

Swelling in the midline, towards the left; moves with deglutition as well as with the protrusion of tongue. Patient is asked to open the mouth and keep the lower jaw still. Examiner holds the cyst between the thumb and forefinger. When patient is asked to protrude the tongue, a 'tugging sensation' can be felt. Swelling is smooth, soft, fluctuant (cystic), nontender, mobile, often transilluminant. Thyroid fossa is empty, if there is no thyroid in normal location. Thyroglossal cyst can get infected and may form an abscess. Malignancy can develop in thyroglossal cyst (papillary carcinoma).

Investigations

Radioisotope study; US neck; FNAC from the cyst; T_3 , T_4 and TSH estimation.

Differential Diagnosis for Thyroglossal Cyst

Subhyoid bursa; pretracheal lymph node; dermoid cyst; solitary nodule thyroid. If tract is not completely excised, it will result in thyroglossal fistula.

Note: Thyroid cartilage is shaped like a prow of a ship and so thyroglossal tract during development sweeps towards one side. So levator glandulae thyroideae in normal people and thyroglossal cyst when develops will be towards left side (**Figs 14.56 to 14.58**).

Thyroglossal Fistula

It is not a congenital condition. It either follows infection of thyroglossal cyst which bursts open or after inadequate removal of the cyst. It is lined by columnar epithelium, discharges mucus and is a seat of recurrent inflammation. 'Hood sign' is characteristic (crescentic appearance is due to uneven rate of growth of thyroglossal tract). It may be located in suprahyoid position or on the side of the old scar (**Fig. 14.59**).

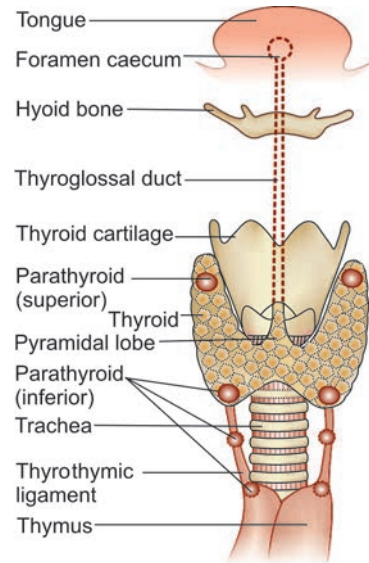


Fig. 14.56: Thyroglossal duct anatomy.

Investigations

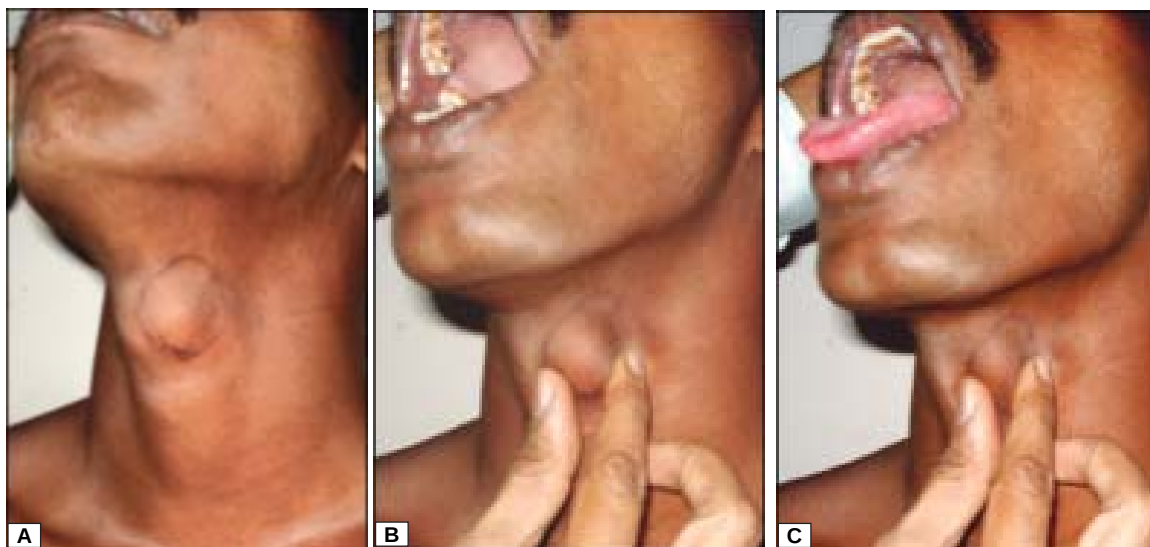
Radioisotope study, study of discharge and fistulogram. It may mimic tuberculous sinus.

Dyshormonogenesis

It is an autosomal recessive condition wherein there is either deficiency of thyroid enzymes (either peroxidase or dehalogenase) or inability to concentrate or to bind or to retain iodine. It may be familial and patient presents with large diffuse vascular goitre involving both lobes. They respond very well to L-thyroxine and may not require surgery at any time. Condition may be associated with congenital deafness which is being called as *Pendred's syndrome*.

Ectopic Thyroid

Ectopic thyroid tissue may lie anywhere along the line of descent of thyroid during the developmental period. Whole of the thyroid gland or residual thyroid lies in an abnormal position either in the posterior part of the tongue, or in the upper part of the neck in midline, or intrathoracic region. Radioisotope scan, CT scan for intrathoracic thyroid will confirm the diagnosis (**Fig. 14.60**).



Figs 14.57A to C: Thyroglossal cyst examination to feel the unmistakable upward 'tug'.



Fig. 14.58: Thyroglossal cyst—suprahyoid position.



Fig. 14.59: Thyroglossal fistula.

Lingual Thyroid

It is a thyroid swelling in the posterior third of tongue, at the foramen caecum, presenting as rounded swelling. It may be the only existing thyroid tissue which may cause dysphagia, speech impairment, respiratory obstruction, haemorrhage. Any diseases which can occur in normal thyroid can also occur in lingual thyroid, i.e. nodularity, toxicity, malignancy.

Diagnosis: Radioisotope study shows the uptake of iodine by the lingual thyroid and also says the status of the thyroid in normal fossa. US neck has to be done to see the absence of thyroid in normal location.

Goitre in Infancy

It is seen in endemic area or in infants whose mother was taking antithyroid drugs for thyrotoxicosis

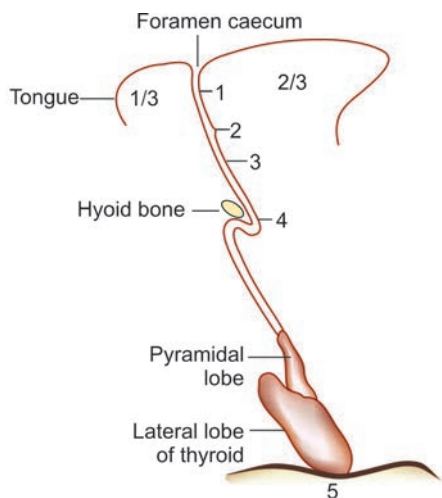


Fig. 14.60: Thyroid ectopic sites. 1: Ectopic lingual thyroid, 2: Intralingual thyroid, 3: Sublingual thyroid, 4: Thyroglossal cyst, 5: Retrosternal thyroid.

(excessive TRH secretion in mother crosses the placenta). Untreated thyrotoxicosis in pregnancy also can cause toxic goitre in infant.

Myxoedema (Word Meaning is Mucous Swelling)

It is a clinical state of severe lack of thyroid hormone. It is common in middle aged and elderly. It is common in females. Tiredness, weakness, mental lethargy, cold intolerance, weight gain, poor appetite, dyspnoea, ankle oedema, slow speech, decreased thinking, menorrhagia are the symptoms. **Signs:** Previous scar of thyroidectomy or nodular goitre or normal neck; swollen heavy eyelids; loss of hairs on the lateral third of eyebrows; smooth, pale yellow creamy skin; flushed pink orange cheeks (peaches); thin ragged hairs; over deposition of fat and connective tissues in supraclavicular fossa, back, neck and shoulders; dry, elastic, nonsweating skin; nonpitting oedema; puffy spade like hands; enlarged tongue; deep and hoarse voice; bradycardia (40-60 beats/minute); low blood pressure; cold hands; blue fingertips; sluggish reflex with prolonged relaxation period. *Myxoedema coma* develops eventually with hypothermia, hypotension, hyponatraemia, hypoventilation, hypoglycaemia, *deadly cold skin* like of a toad; rectal temperature below 24°C.

Hyperparathyroidism

Types

(1) *Primary*. (2) *Secondary*. (3) *Tertiary*. **Primary** is unstimulated inappropriate high PTH secretion due to hyperplasia or adenoma. **Secondary** is due to chronic renal failure or due to malabsorption, i.e. due to chronic hypocalcaemia. **Tertiary** is due to autonomous reactive parathyroid hyperplasia seen after renal transplantation.

Primary Hyperparathyroidism

Adenoma—75%; Hyperplasia—20-24%; Carcinoma—rare—1%. Carcinoma of parathyroid is one of the most aggressive tumour known but fortunately rare. May present as a nodule and can have blood born metastasis.

Clinical features: Clinical vignette of hyperparathyroidism—**“Bones, stones, abdominal groans and psychic moans.”** (1) Hyperparathyroidism is common in middle aged women. (2) Presentation may be *asymptomatic in 50% cases*. (3) *Nonspecific symptoms and psychiatric symptom* (They are most often named as neurotics). (4) Behavioural problems. (5) In the bone there will be *osteitis fibrosa cystica (von Recklinghausen disease of bone)* which shows single or multiple cysts or pseudotumour in the jaw, skull or middle phalanges. (6) Lamina dura of tooth is the first bone to show changes. (7) In the kidney there may be bilateral multiple *renal stones or nephrocalcinosis* (may go for renal failure). (8) It may be associated with the *peptic ulcer, pancreatitis, MEN I syndrome*. (9) They are more prone for skin necrosis, band keratopathy, pseudogout, myalgia, arthralgia, polyuria, glycosuria and hypertension. **Acute hyperparathyroidism crisis:** It is rare but dangerous presentation (crisis) wherein patient presents with abdominal pain, vomiting, dehydration, oliguria and death. Serum calcium is very high.

Investigations: High serum calcium - > 10 mg/100 ml; Decreased serum phosphorus; Increased urinary calcium - > 250 mg/24 hrs; Increased serum alkaline phosphatase; Increased PTH level in the serum is diagnostic - > 0.5pg/L; X-ray skull shows *salt-pepper appearance*; X-ray phalanges and jaw is specific; US abdomen to find out problems in kidney, pancreas;

US neck or CT scan neck and mediastinum may show the parathyroid adenoma; Selective venous sampling for PTH is also very useful; Thallium—Technetium scan shows *hot spots* which is diagnostic of parathyroid adenoma; ^{99m}Tc Technetium labeled Sestamibi isotope scan is more sensitive.

Differential diagnosis: Sarcoidosis; over intake of vitamin D; secondaries in bone; multiple myeloma; functioning carcinomas.

MEN Syndrome (MEA Syndrome)

It is multiple endocrine neoplasia syndrome. It is commonly inherited as *autosomal dominant*; Cells involved has got common features of *apud cells* (*Apudomas*).

Types: **Type I:** Parathyroid hyperplasia or adenomas; pituitary tumour; pancreatic tumour [Endocrine- (Insulinoma, gastrinoma, glucagonoma, vipoma)]. It is also called as *Wermer's syndrome*. **Type II:** Also called as *Sipple's disease*; *IIA* includes medullary carcinoma of thyroid + phaeochromocytoma + parathyroid hyperplasia (50%); *IIB* includes medullary carcinoma of thyroid + phaeochromocytoma + mucosal neuromas in lips and eyelids with bumpy-lumpy lesions, with marfanoid face, megacolon.

Tetany

It is decreased level of calcium in blood causing its effects.

Causes: After thyroidectomy (it is decreased level of parathormone in the blood causing hypocalcaemia). It is usually temporary lasts for 4-6 weeks. It is the commonest cause of hypoparathyroidism. Other causes of hypoparathyroidism are neck dissection, haemochromatosis, Wilson's disease, di-George's syndrome (absence of parathyroids; thymic aplasia; cardiac



Fig. 14.61: Carpal spasm due to tetany in post-thyroidectomy patient. Note the thyroidectomy scar.

defects); severe vomiting, hyperventilation associated with respiratory alkalosis; metabolic alkalosis; rickets, osteomalacia; chronic renal failure; acute pancreatitis.

Clinical features: *Decreased PTH* causes decrease in calcium level in the blood leading to *circumoral paraesthesia*, paraesthesia of neck, fingers and toes, Twitching and weakness of tongue muscles, muscles of forearm, hand, foot and digits—*carpopedal spasm*; *Chvostek-Weiss's sign*—tapping above the angle of the jaw stimulates branches of facial nerve causing the twitching of the angle of mouth and eyelids; Applying the sphygmomanometer to the arm and inflating the pressure more than systolic pressure of the patient for three minutes can demonstrate carpal spasm (*Trousseau's sign*); *Stridor* and difficulty in breathing due to paralysis of respiratory muscles; Generalized weakness and twitching all over the body in severe cases mimicking *convulsions* (**Fig. 14.61**).

15

Examinations of Face and Head

Introduction

Many specific and peculiar conditions pertaining to face and head occurs. Because of their individuality, they are being discussed as a separate chapter. Relevant history, examination methods are same as in chapters—ulcer, swelling, neck and oral cavity. Student should refer specific chapters for method of examination (**Figs 15.1 to 15.4**).

Hippocratic facies: It is seen in patients with acute severe peritonitis with terminal illness. Features are—sunken bright eyes, pinched nose, dry, shriveled tongue, crusted lips, cold clammy forehead, distended abdomen with features of peritonitis.

Adenoid facies: High vaulted palate, narrow dental arch, protruding incisor teeth, earlier was considered as feature of enlarged adenoid is now not accepted. In fact these features are familial anomaly. Enlarged adenoids are a coincidental.



Fig. 15.1: Conjunctival haemorrhage.

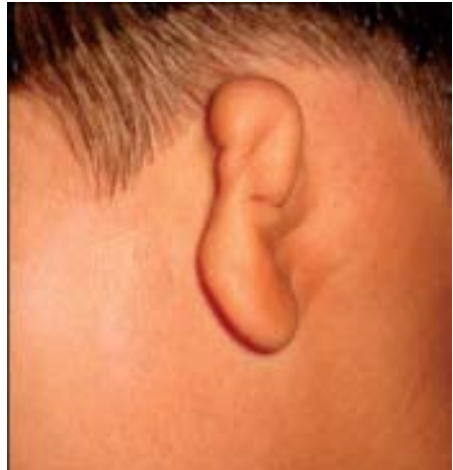


Fig. 15.2: Underdeveloped ear—anomaly.



Fig. 15.3: Developmental anomaly of face with underdeveloped mandible.



Fig. 15.4: Accessory ear. An accessory auricle is a protruded posterolateral part of the face with cartilage, fibrous tissue and skin covering. It is commonly cylindrical erect in front of the tragus. It is sequestration of an island of cartilage from the mandibular arch during closure of first branchial cleft.

Facies of cretinism—seen in infancy; pale, puffy, wrinkled face; dry cold skin; protruded tongue; open anterior fontanelle; palpable (in endemic type) or impalpable (in sporadic type gland is atrophic) thyroid gland.

Facies of congenital syphilis: Bossing of frontal bones; interstitial keratitis; Hutchinson's teeth; saddle nose.

Facies of hepatic cirrhosis: Sunken eyes; jaundiced sclera; watery conjunctiva.

Virile facies in a women suffering from adrenocortical hyperplasia or tumour is typical (face looks like that of men).

Moon face of Cushing's syndrome: Rubicund round face like of full moon; pursed lips.

Face of myasthenia gravis: Unilateral or bilateral intermittent ptosis; drooping jaw; sneering smile face due to reduced action of risorius and zygomatic muscles.

Carcinoid facies: Typical facial flushing seen in metastatic carcinoid tumour.

Rhesus sardonius face of tetanus with trismus—painful smiling.

Cleft Lip and Cleft Palate

Development of Face

Face develops from median nasal process, lateral nasal process, maxillary process, mandibular arch, globular arch, olfactory pit and eye. Any change in the development or fusion of these arches leads to formation of different types of cleft lip or cleft palate.

Aetiology

Familial—More common in cleft lip or combined cleft lip and palate (Risk is 1:25 live births); protein and vitamin deficiency; Rubella infection; radiation; chromosomal abnormalities; maternal epilepsy and drug intake during pregnancy (steroids/eptoin/diazepam).

Classification

- I. *Cleft lip alone*: Unilateral; Bilateral; Median.
- II. *Cleft of primary palate (in front of incisive foramen) only*: (a) Complete—means absence of pre-maxilla. (b) Incomplete—means rudimentary pre-maxilla: Unilateral; Bilateral; Median.
- III. *Cleft of secondary palate (behind the incisive foramen) only*: a) Complete – nasal septum and vomer are separated from palatine process. b) Incomplete. c) Submucous. It can be – Cleft with soft palate involvement. Cleft without soft palate involvement.
- IV. *Cleft of both primary and secondary palates*.
- V. *Cleft lip and cleft palate together*.

Defect is often associated with other congenital anomalies of cardiac, gastrointestinal, neurological system, *Pierre-Robin syndrome* (most commonly associated syndrome with features of isolated cleft palate, retrognathia, posteriorly displaced tongue), *Klippel-Feil syndrome*, *Stickler's syndrome* (eye, skeletal, muscular, cleft), *Shprintzen's syndrome* (cardiac and cleft disorder), *Down's syndrome*, *Treacher-Collin's syndrome*, *Apert's syndrome* and trisomy.

Incidence

Common in Caucasians; in 75% of cases it is unilateral. Commonly occurs on the left side (60%); in 50% of

cases it is combined cleft lip and palate. Incidence is 1:600 live births; common in boys; in 15-25% of cases it is cleft lip alone; in 25-40% of cases it is cleft palate alone. Incidence is 1:1000 live births; more common in girls.

Problems in Cleft Disorders

Difficulty in sucking and swallowing. This is commonly observed in cleft palate than in cleft lip; speech is defective especially in cleft palate, mainly to phonate B, D, K, P, T and G. Altered dentition or supernumerary teeth; recurrent upper respiratory tract infection; respiratory obstruction (in Pierre-Robin syndrome); chronic otitis media, middle ear problems; cosmetic problems; hypoplasia of the maxilla; problems due to other associated disorders (Figs 15.5 to 15.9).

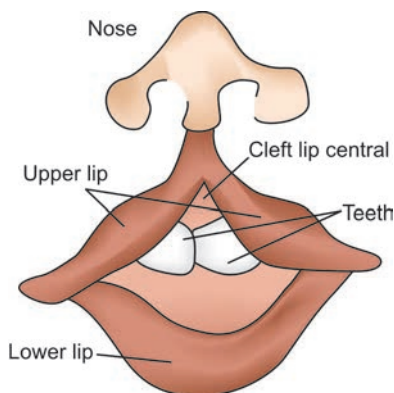


Fig. 15.5: Central cleft lip (Hare lip, Type I cleft lip—It is rare).

Cleft lip

- *Central*—Rare. In upper lip. Between two median nasal processes. (*Hare lip*)
- *Lateral*—Maxillary and median nasal process, commonest; can be unilateral or bilateral
- *Incomplete* cleft lip does not extend into nose
- *Complete* cleft lip extends into nasal floor
- *Simple* cleft lip is only cleft in the lip
- *Compound* cleft lip is cleft lip with cleft of alveolus

LAHS classification of cleft disorders

- 'L' for lip, 'A' for alveolus, 'H' for hard palate, 'S' for soft palate
- Capital 'LAHS' for 'complete' type
- Small letters 'lahs' for 'incomplete type'
- Asterisks 'lahs' for microclefts
- 'LAHSHAL' for bilateral clefts

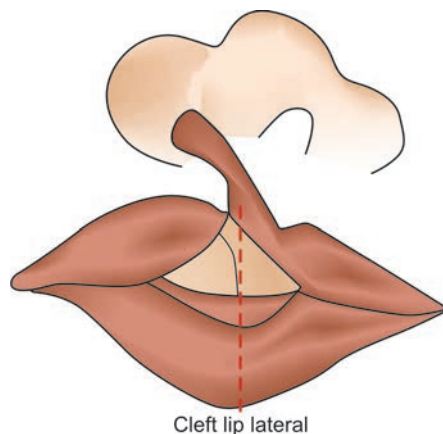


Fig. 15.6: Lateral type of cleft lip (Type II variety—it is commonest). It is due to imperfect fusion of maxillary process and median nasal process. It can be unilateral or bilateral.



Fig. 15.7: Bilateral cleft lip.

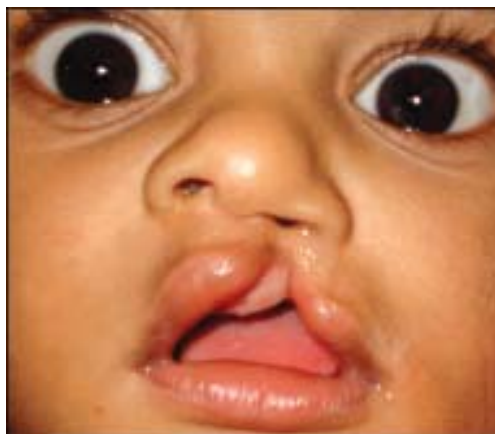


Fig. 15.8: Unilateral cleft lip, lateral type which is commonest.



Fig. 15.9: Bilateral cleft lip involving palate.

Cleft Palate

It is due to failure of fusion of the two palatine processes; defect in fusion of lines between premaxilla (developed from median nasal process) and palatine processes of maxilla one on each side; when premaxilla and both palatine processes do not fuse, it leads into complete cleft palate (Type I cleft palate). Incomplete fusion of these three components can cause incomplete cleft palate beginning from uvula towards posteriorly at various lengths. So it could be Type IIa—Bifid uvula, Type IIb—bifid soft palate (entire length) or Type IIc—bifid soft palate and posterior part of hard palate (but anterior part of hard palate is normal). Small maxilla with crowded teeth, absent/poorly developed upper lateral incisors. Bacterial contamination of upper respiratory tract with recurrent infection is common. Chronic otitis media with deafness may occur. Swallowing difficulties to certain extent and speech problems can occur; cosmetic problems can occur (**Figs 15.10 to 15.14**).

Bifid Nose

One-half of the frontonasal process remains isolated from rest.

Facial Cleft

Lateral nasal process fails to unite with maxillary process causing a fissure from upper lip to the inner canthus of the eye alongside of the nose.

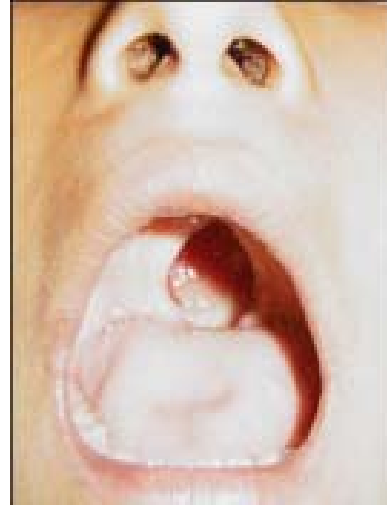


Fig. 15.10: Cleft palate only. Lip is normal. Premaxilla is not involved.

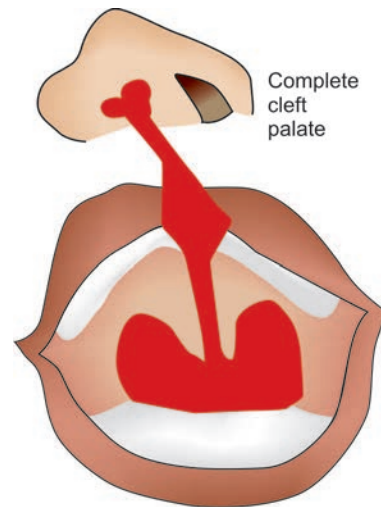


Fig. 15.11: Complete cleft palate Type I.

Macrostoma

Size of the mouth is more than the normal due to imperfect union of maxillary process with mandibular arch.

Mandibular Cleft

Mandibular arch of one side fails to unite with mandibular arch of opposite side.

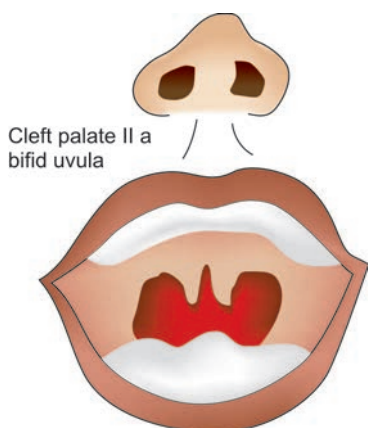


Fig. 15.12: Cleft palate Type II a—bifid uvula.

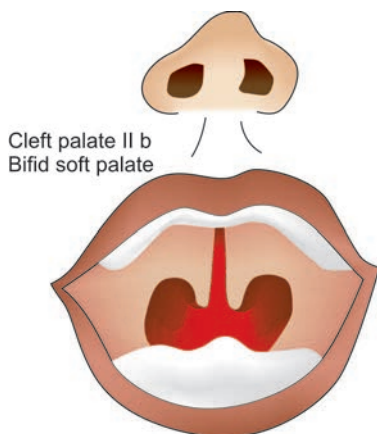


Fig. 15.13: Cleft palate Type II b—bifid soft palate (entire soft palate).

Cleft palate II c—bifid soft palate and Cleft post hard palate

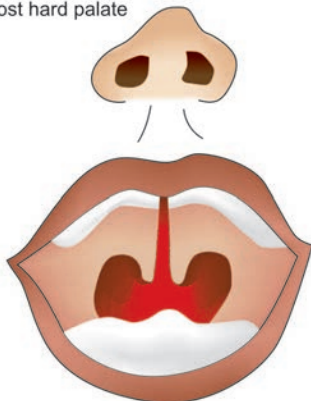


Fig. 15.14: Cleft palate Type II c—bifid soft palate entire length with cleft of posterior hard palate (anterior palate is normal).

Congenital Short Frenum of Upper Lip

It is seen with a wide gap between the permanent incisor teeth. Congenital fistulae of lower lip are two rare blind pits one on either side of the midline containing wide open mucus secreting glands.

Hydrocephalous

It is dilatation of ventricles due to blockage of flow of cerebrospinal fluid (CSF).

Classification I

(a) *Communicating type*: Ventricles communicate freely into the subarachnoid space. Here there is defective absorption of CSF following any inflammation, subarachnoid haemorrhage or trauma. (b) *Noncommunicating type*: Obstruction is in the ventricle or its exit due to any tumours or any inflammatory process.

Classification II

Congenital: It is associated with spina bifida/myelomeningocele. There is failure of formation of CSF pathway. It is associated with *Arnold-Chiari syndrome*, congenital stenosis of aqueduct of Sylvius. **Clinical features**: Widening/separation of suture lines; bulged tense fontanelle; engorged scalp veins; sun setting eye; decreased cortical thickness; enlarged head. **Acquired**: It may be unilateral or bilateral. It is due to chronic meningitis, trauma, subarachnoid haemorrhage, brain tumours, colloid cyst of 3rd ventricle, arachnoid cysts (**Fig. 15.15**).



Fig. 15.15: Hydrocephalous.

Meningocele

Meningocele is protrusion of the meninges. It contains clear fluid—CSF. It is brilliantly transilluminant. It shows impulse on coughing or crying. **Meningo-encephalocele** is protrusion of brain also along with meninges. It is transilluminant. **Encephalocele** is protrusion of brain. It is not transilluminant. There may be neurological deficits, incontinence of urine and faeces. These conditions are seen in midline – root of the nose, occiput, anterior fontanelle region. Often it is associated with spina bifida (**Fig. 15.16**).



Fig. 15.16: Encephalocele.

Preauricular Sinus

It is due to failure of fusion of anterior tubercles of the auricle creating a sinus. Ear develops from six tubercles. This sinus opens at the root of the helix or on tragus. Sinus track runs downwards and ends blindly. Often sinus opening gets sealed forming a preauricular cyst which gets infected forming an abscess. Sinus can get infected repeatedly discharging pus through its opening. It is often multiple. Sinusogram and study of discharge is needed. It often mimics cold abscess or sebaceous cyst (**Fig. 15.17**).

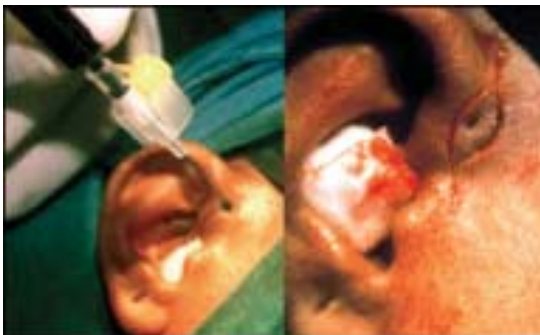


Fig. 15.17: Preauricular sinus.

Traumatic Problems of Face and Head

Head, faciomaxillary injuries are discussed in detail in Chapters 11: *Examination of Jaw* and 29: *Examination of Intracranial Diseases*.

Haematoma scalp is very common traumatic swelling observed. It is common in 2nd layer – connective tissue dense or 4th layer galea aponeurotica. Haematoma in 2nd layer is localised, tender and tense swelling. Haematoma in the 4th layer is often diffuse and extensive. In front it may extend into the root of nose and eyelids as galea aponeurosis is not attached to any bone in front. Fracture of underlying skull bone should be also thought of. Neurological deficits should be assessed using Glasgow coma scale. Fracture in the line of venous sinuses can be dangerous and life threatening.

Subperiosteal haematoma also called as *cephal-haematoma*. It is collection of blood under the pericranium. It is common in newborn after forceps delivery. It is common in parietal region. It is localised, smooth, soft, fluctuant swelling limited to the suture lines of the particular bone. It gradually disappears in few months (**Figs 15.18 and 15.19**).

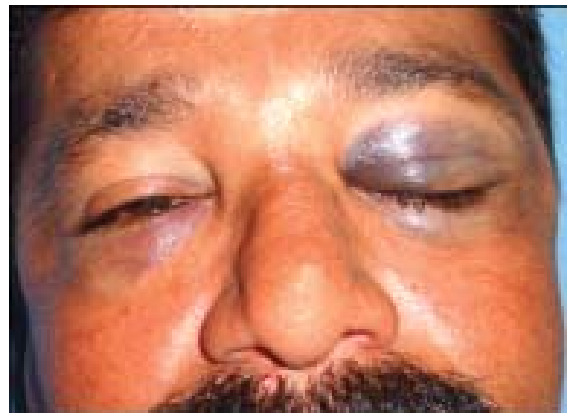


Fig. 15.18: Traumatic eyelid haematoma.

Problems with Infective Lesions of Face and Head

Cavernous Sinus Thrombosis

Infection from face and scalp may extend through various routes to cavernous sinus causing its thrombosis. Routes are – along angular vein to ophthalmic vein; along pterygoid plexus of veins which



Fig. 15.19: Extensive soft tissue injury of face.

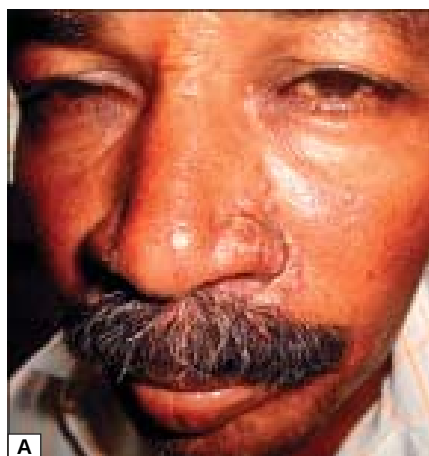
communicate deep facial vein to cavernous sinus across foramen ovale and foramen lacerum. Patient develops toxicity, proptosis, squint, ocular muscle paralysis specifically lateral rectus which is supplied by abducent nerve which is situated within the cavernous sinus. *Dangerous zone* in the face is located in the area of nose and upper lip as infection in this area is more prone to develop cavernous sinus thrombosis. Any boil, cellulitis, erysipelas, abscess in this zone can cause this complication (**Figs 15.20A and B**).

Pott's Puffy Tumour

Pott's puffy tumour is localised pitting oedema of the scalp with adjacent cranial bone osteomyelitis. Acute pain, localised swelling and tenderness with often osteomyelitis of the underlying bone are the features. It is common in frontal region. Acute frontal sinusitis may be the initial pathology. Intracranial spread may cause extradural abscess.

Cancrum Oris

Cancrum oris is an infective gangrenous stomatitis destructing gums, gingivae, cheek; seen in debilitated children after measles, kala azar, typhoid (**Fig. 15.21**).



Figs 15.20A and B: Dangerous area – triangular area in the nose and upper lip.



Fig. 15.21: Cancrum oris extensively involving gingiva and lower lip with plenty of maggots in it.

Lupus Vulgaris

Lupus vulgaris is cutaneous tuberculosis extensively involving face often, with destruction and 'apple jelly' like lesions.

Actinomycosis of Mandible

Actinomycosis of mandible can cause multiple sinuses discharging sulphur granules in the lower jaw. Microscopy shows 'Ray fungus' nature of the bacteria *Actinomycosis israelii*.

Benign Swellings of the Face and Head

Papilloma, lipoma, haemangioma, sebaceous cyst, Cock's peculiar tumour due to sebaceous cyst, dermoid cyst, osteoma, cirroid aneurysm, mucus cyst of lips can occur (Figs 15.22 to 15.25).



Fig. 15.22: Dermoid ear.



Fig. 15.23: Eyelid swelling—upper eyelid.



Fig. 15.24: Multiple scalp swellings (multiple sebaceous cysts) scalp.



Fig. 15.25: Sebaceous horn scalp.

Cirroid Aneurysm

Cirroid aneurysm is seen only in face in the forehead affecting the superficial temporal artery, as dilated interwoven artery and its branches. It feels like a 'bag of pulsating earthworms' with thinned out overlying skin with loss of hair ; often ulceration and severe bleeding can occur. Intracranial extension is known to occur into extradural space. X-ray skull shows bone erosion.

Osteoma

Osteoma is common in skull bone which is compact or ivory type and is sessile type. It affects the outer table of the skull bone – frontal, parietal or occipital bones. Painless bony hard nonmobile swelling in the skull bone is the presentation. It does not turn into malignancy.

Paget's Disease of Bone

Paget's disease of bone causes progressive enlargement of the skull with thickened skull bones with systolic bruit on auscultation due to vascularity.

Malignant Conditions of Face and Head

Malignant conditions like basal cell carcinoma (*rodent ulcer*), squamous cell carcinoma of lip or skin, malignant melanoma, secondaries in skull, osteosarcoma, can occur. Secondaries in skull can occur from primaries from thyroid, kidney, lungs, adrenals, breast, etc. It is hard tender, multiple. It can be solitary also. Soft, localised warm vascular pulsatile secondaries are seen in secondaries from follicular carcinoma of thyroid.

Cylindroma

Cylindroma often called as *turban tumour* occurs in the scalp involving entire scalp area as red lobulated slow growing relentless rare tumour which is locally malignant with alopecia in the affected area. It should be differentiated from plexiform neurofibromatosis and temporal arteritis.

Examination of Cranial Nerves

Cranial nerve palsies that commonly presents in head and face is being discussed in this chapter.

Cranial nerves are Olfactory; Optic; Oculomotor; Trochlear; Trigeminal; Abducent; Facial; Auditory; Glossopharyngeal; Vagus; Accessory; Hypoglossal nerves. (Mnemonic—On Old Olympus Towering Tops A Finn And German Picked Some Hops).

Olfactory

Sense of smell is tested with cloves, peppermint, etc.

Optic

Visual acuity (ability to read), visual fields (peripheral vision to be checked in one eye and compared to examiner's), Colour vision using charts.

Oculomotor

It supplies all extrinsic muscles of eyeball except superior oblique (trochlear), lateral rectus (abducent),

levator palpebrae superioris and muscle of accommodation. In oculomotor nerve palsy eye looks downwards and outwards with ptosis (drooping of upper eyelid) and fixed pupil. Superior rectus - to look up; medial rectus - to converge; inferior rectus - to look down; inferior oblique - to look up and out.

Trochlear

It supplies superior oblique muscle. When it gets damaged turning eye downwards and outwards is defective and patient looks inwards with diplopia below the horizontal line.

Trigeminal

Sensory supply is to entire one side of the face by three divisions - ophthalmic - upper; maxillary - middle; mandibular - lower. Ophthalmic division also supplies conjunctiva. Maxillary branch supplies mucous membrane of nose, pharynx, roof of mouth, soft palate and tonsil; mandibular division to tongue, lower teeth, mucous membrane of the mandible. Sensations should be checked in this place. Conjunctival reflex, palatal reflex will be altered. In trigeminal neuralgia there is hyperaesthesia with touch becoming pain. During the period of neuralgic attack entire area is hyperaesthetic. *Only certain trigger zones of Patrick* are hyperaesthetic in between attacks. Motor supply to masseter, pterygoids and temporalis is from mandibular branch. Clenching the teeth will confirm the same. While opening the mouth widely jaw deviates towards the affected side due to weakness of pterygoids. Taste from anterior 2/3rd is through lingual nerve via chorda tympani from geniculate ganglion. Sweet (sugar), sour (acid), salt (salt) and bitter (quinine) tastes are checked. Salt and sweet in the tip of the tongue (through chorda tympani); sour is in lateral margin of tongue through trigeminal nerve; bitter is in posterior tongue through glossopharyngeal nerve.

Abducent

Abducent nerve supplies the lateral rectus muscle of the eye. Turning of eye outwards is defective in its paralysis and attempt to look sideways will cause diplopia.

Facial

It supplies muscles of facial expression. It is motor nerve. Supranuclear palsy causes lower facial palsy;

infratemporal palsy causes entire facial nerve palsy. Features include—Eyelids cannot be closed; whistling is defective; angle of the mouth deviates; wasting of the muscles of the side; wrinkling of eye is defective; inability to close the eyes properly (**Fig. 15.26**).

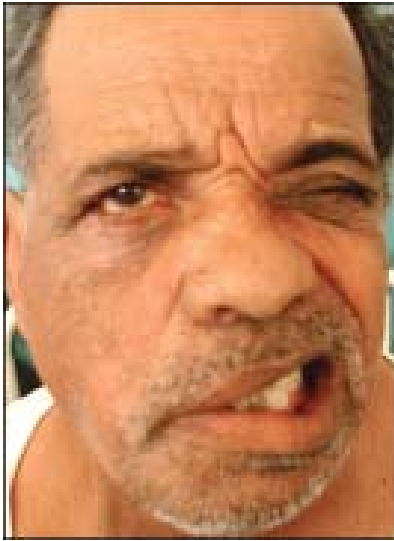


Fig. 15.26: Features of facial palsy.

Auditory

It supplies cochlea and semicircular canals. *Weber's tuning fork test* is used to rule out conductive deafness. After placing the tuning fork on the forehead louder sound is felt on the side of conductive deafness. Tuning fork is placed on mastoid to get louder sound in conductive deafness in *Rinne's test*. In sensory deafness there is no change in sound appreciation. Assessing

the response to changes in temperature in the external meatus – *calorie test* is used to check the sensitivity of the vestibular apparatus.

Glossopharyngeal

It is sensory to posterior third of the tongue (and also carries bitter taste checked by using quinine) and to mucous membrane of pharynx. It is motor to middle constrictor. *Gag reflex* can be elicited by stroking the back of oropharynx.

Vagus

It is motor to soft palate, pharynx and larynx and sensory to gut, heart and lungs. After opening the mouth patient is asked to say 'Aahh'. Soft palate arches upwards symmetrically. In paralysis of one side, it will not arch symmetrically and uvula gets pulled towards functioning (*opposite*) side. Change in voice, inability to cough and vocal cord palsy in indirect laryngoscopy are the other features.

Spinal Accessory

Wasting of sternomastoid and trapezius is obvious. When chin is pushed towards opposite side against resistance weakness can be appreciated; shrugging of the shoulder against resistance when checked from behind is defective.

Hypoglossal

It is motor to tongue. When it is paralysed, wasting of tongue is seen on the same side; tongue deviates towards *same side* while protruding out.

16

Examination of Breast

Pain and/or lump in the breast are the common complaints for which patient consults a surgeon or gynaecologist or breast clinic.

History

Chief Complaints

- Swelling in the right/left breast/both breasts; its time duration.
- Pain in the breast with duration; ulceration in the breast with duration.
- Discharge from nipple.
- Swelling in the breast/axilla/neck.

History of Present Illness

Swelling: History of duration of swelling, its progression whether slowly increasing in size or rapidly increasing has to be asked for. Swellings of short duration are most probably due to carcinoma. But most often, once the swelling is noticed the patient immediately consults a doctor for opinion and so duration may not be clearly obtained. Condition like fibroadenoma and fibroadenosis has got long duration of history. Duration in carcinoma is usually only few weeks. History of swelling in the opposite breast is also important. In 2% of cases, breast carcinomas are bilateral; and so also fibrocystadenosis which commonly has bilateral presentation.

Pain: Pain in the breast is often termed as *mastalgia*. It is common in fibrocystadenosis and acute mastitis. There will be associated fever in mastitis. Carcinoma breast is initially painless but eventually becomes painful following infiltration or development of tumour necrosis or skin ulceration/fungation. Pain in fibroadenosis is more prior to menstruation (cyclical), and may

disappear during pregnancy and after menopause. Duration of pain, type, timing, site and relation to menstruation has to be noted. Referred pain from muscle and skeletal system (ribs) can also develop in the breast. Periductal mastitis/duct ectasia can cause pain. Patient with breast abscess will show severe excruciating pain in the breast.

Nipple discharge: Duration of discharge, its type whether it is of serous/purulent/bloody/serosanguinous/milky/greenish type has to be asked for and noted. Bloody discharge is often seen in duct papilloma, carcinoma. Serous and greenish discharge is seen in fibroadenosis.

History of changes in nipple: Like retraction (depression), deviation, destruction, displacement, discolouration, duplication and discharge is noted. Recent history of changes signifies carcinoma. Often retraction may be congenital, since birth.

History of alteration in size and asymmetry of the breasts should be asked for with duration.

History of trauma: Trauma may cause haematoma in the breast and breast abscess. Direct or indirect trauma often can cause traumatic fat necrosis after few weeks. Here trauma may be forgotten or may not be noticed by the patient and swelling developed due to traumatic fat necrosis is painless, nonprogressive and nonregressive.

History related to swelling in the axilla/neck and their details like duration, progress, pain, ulceration, etc. is noted.

History related to respiratory problems has to be asked like chest pain/breathlessness/cough/haemoptysis—signifies the secondaries in lung from carcinoma breast.

History of abdominal pain, loss of appetite, decreased weight, jaundice, and abdominal distension should be asked for which signifies liver secondaries.

History related to bone secondaries—like bone pain, low back pain, altered sensation like sense of position and vibration, lower limb weakness, features of paraplegia, loss of control over urination and defecation is asked for.

History of convulsions, loss of consciousness, vomiting, limb weakness, headache, visual disturbances, behavioral changes (psychological changes) and localisation changes may be seen whenever there is brain metastases.

Past History

Past history of any surgeries of breast (recurrence can occur after excision of fibroadenoma, conservative breast surgery may cause recurrent carcinoma breast) or drug therapies like for fibroadenosis. Abscess may recur in congenital retraction of nipple; tuberculosis of breast can show recurrence; fibroadenosis may present repeatedly with long gaps of asymptomatic period.

Menstrual History, Obstetric History and Family History

This is important in breast diseases as breast carcinoma can be familial. Family history of carcinoma of breast (in mother, grandmother, aunt, cousins, and 1st and 2nd degree relatives), ovarian tumour or other tumours has to be noted. Often multiple tumours can occur. History of age of menarche and menopause, menstrual cycles, marital status, number of pregnancies, breast-feeding, last child birth and usage of contraceptives/postmenopausal HRT are very important. Fibroadenosis and carcinoma are more common in unmarried individuals.

Personal History and Treatment History

History of smoking, alcohol intake, dietary habits (high fat diet) is noted. History of any drug intake at present is important.

General Examination

Like for any other long case, patient should be examined for pallor, jaundice, oedema feet and clubbing. Pulse and blood pressure should be checked.

Local Examination of Breasts

Usually normal breast should be examined first. Proper exposure of both breasts from neck to waist should be done. While examining the breasts adequate privacy; and presence of a female nurse is a must. Initially examination is carried out with the patient sitting in *45° semi-recumbent position* (lying flat makes breasts flatten and fall sideways; upright sitting position makes breasts pendulous and bulky). Later examination is done in lying down (*recumbent*) position as lump is better felt against chest wall for additional information. During inspection, the clinician should stand in front and later on the side of the patient. *Commonly used position is sitting posture* as it is easier to examine nipples, lump and axillary nodes; and patient also will be more comfortable in that position.

Breast is examined in different positions to elicit different clinical features.

Different positions are-

- Sitting position with arms by the side
- *45° semi-recumbent position* is very much convenient
- Sitting position with leaning forward
- Sitting position with arms over the waist
- Sitting position with arms rising above the shoulder to see fixity to chest wall and changes in nipple
- Lying down position for self-examination

Inspection

For proper inspection, both breasts should be exposed properly including axillae. Inspection is done in sitting position with the arms by the side of the body. Inspection is also done with the arms raised above the shoulder touching the head (with arms touching the ears) so that nipple levels, lump, dimples are seen well. Inspection is also done with the arms on the hips pressing and relaxing so that skin dimpling, nipple movements and changes become more prominent. Examination/inspection done in *bending forward* position helps to see whether breast falls forward or not; and also to see nipple retraction or failure of nipple to fall away. Carcinoma fixed to chest wall will not fall forward while bending forward (**Figs 16.1A to 16.3**).



Figs 16.1A to C: Examination of breast is done in sitting position with arms beside.



Figs 16.2A to C: Examination with both arms raised above the shoulder and leaning forward.



Fig. 16.3: Examination in 45° semi-recumbent position.

Inspect both breasts—note the size, shape and symmetry. Asymmetry can be seen in breast lumps. Inspect both breasts while leaning forward to see whether both breasts fall forward or not. In carcinoma, if the breast lump gets fixed to underlying chest wall, it will not fall forward. Both breasts should be inspected while the arms are raised upwards to see whether breast is/breasts are adherent to chest wall (**Fig. 16.4**).

Inspection of nipple—Look for symmetry/asymmetry, pushed up/down, displacement, retraction, size/shape

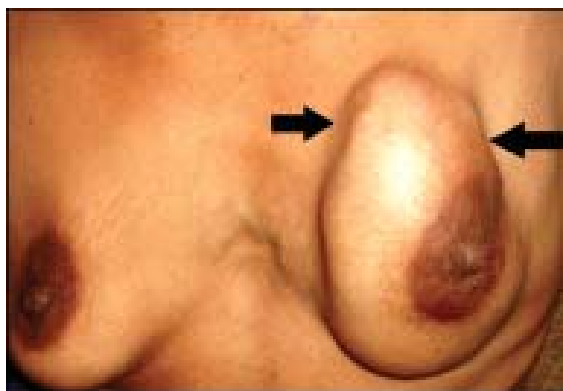
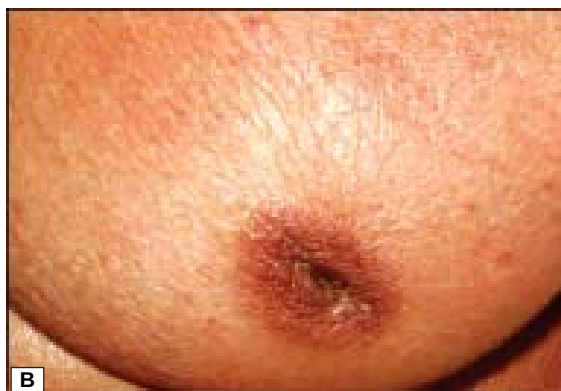
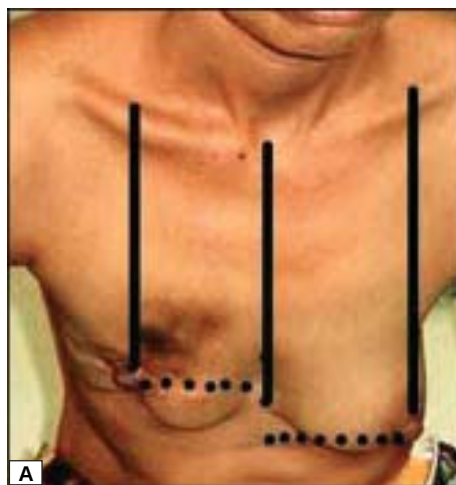


Fig. 16.4: Lump in the breast left sided. Obvious lump is visible in the upper quadrant.

of nipple, discharge/ulceration in the nipple, discolouration, duplication, cracks/fissures. Many of these changes occur in carcinoma. Fissuring and cracks can occur in breastfeeding mothers. Nipple retraction of recent onset may be due to infiltration of lactiferous duct by carcinoma. Often congenital retraction may be present; so duration of nipple retraction is very important. Retraction of nipple can occur in duct ectasia/periductal mastitis also. Nipple retraction is circumferential in carcinoma; slit like in periductal mastitis. Vertical distance from the clavicle and horizontal distance from the midline should be measured and compared to opposite side. Nipple may be drawn towards the lump in the affected breast. Nipple elevation may become prominent by raising the arm above the head; which may be due to inflammatory pathology (**Figs 16.5A and B**). In fibroadenoma nipple



Figs 16.5A and B: Nipple deviation and retraction should be looked for in breast lumps.



Fig. 16.6: Bloody discharge from the nipple. Duct papilloma is the commonest cause.

gets displaced away from the lump. Nipple destruction is seen in Paget's disease and fungating/ulcerating carcinoma. Accessory nipple often may be present along the milk line from axilla to groin or in the thigh; which may show milky discharge during lactation. Nipple may become prominent when there is a swelling underneath like cyst/benign tumour/inflammatory oedema. Nipple may be swollen in infection or carcinoma. It is important to note the type of discharge from the nipple – blood, milk, greenish fluid, serosanguinous, purulent. Bloody discharge may be a feature of duct papilloma or carcinoma (**Fig. 16.6**).

Discharges from the nipple

Blood

- Papilloma – commonest cause
- Ectasia
- Carcinoma – 5% of causes for discharge

Serous

- Fibrocystic disease
- Ectasia

Greenish

- Ectasia
- Fibrocystic disease

Purulent

- Infection
- Sometimes malignancy

Milk

- Lactation (Physiological discharge)
- Galactorrhoea

Serosanguinous

- Carcinoma
- Infection

Inspection of the areola: Areola should be inspected for any changes in colour, size, ulceration, eczema/eczema-like changes. Both areolas should be inspected. Areola is pink in colour in young girls, dark coloured in adults, brownish during pregnancy and lactation. Ulceration of nipple can occur in carcinoma and *Paget's disease of breast*, a localised type of carcinoma breast. It should be differentiated from eczema. Eczema is commonly bilateral without any nodule underneath, associated with itching and vesicles, with normal nipple. It is common during lactation. Paget's disease of breast is unilateral, without vesicles and itching, with a hard lump underneath, often with destruction of nipple. Areola may increase in size significantly in soft fibroadenoma or sarcoma; may be shrunken in size in scirrhous carcinoma. In normal individual, areola is slightly corrugated, with Montgomery's glands on it as small nodules. These glands get hypertrophied during pregnancy and lactation to form Montgomery's tubercles. Retention cyst of this gland presenting as smooth, localised soft fluctuant swelling in the areola is known to occur which often may get infected.

Inspection of the skin over the breast: Skin over the breast is inspected for retraction, pigmentation, redness/shining, dimpling, puckering, *peau d'orange*, nodules, ulceration, fungation, and scar. Any dilated veins over the skin and *cancer en cuirasse* is looked for. Involvement/infiltration of the ligament of Cooper by carcinoma causes dimpling (is a small depression) and puckering (a small fold/wrinkle) of skin over the breast. Normal elastic ligament of Cooper becomes inelastic and shorter in carcinomatous infiltration (Dimpling and puckering are inspeactory findings whereas tethering is a palpatory finding). Oedema of skin is due to blockade of cutaneous lymphatics causing burial of sweat glands and hair follicles giving the appearance of orange peel (*peau d'orange*) (**Figs 16.7A and B**). When ulcer is present, its position, size, shape, margin, floor, edge should be noted. *Cancer en-cuirasse* is extensive involvement of the skin over the breast and chest wall with multiple nodules and ulceration by the carcinoma (**Figs 16.8 and 16.9**). It looks like armor coat. Red, oedematous skin is seen in acute mastitis. *Dilated veins* are commonly observed in cystosarcoma phylloides, large breast abscess, and



A



B

Figs 16.7A and B: Peau de orange appearance of skin in carcinoma breast. It is due to blockade of dermal lymphatics.

sarcoma and often in aggressive carcinomas. *Mondor's disease* is superficial thrombophlebitis of veins over chest wall and breast seen in females. It is painful, tender cord-like lesion which on raising the arm above the shoulder causes puckering of skin adjacent to the dilated vein. It is a self limiting disease. Nodules are usually due to carcinoma; often it may be metastatic from the underlying carcinoma breast. Ulceration is due to carcinomatous infiltration of skin. In cystosarcoma phylloides and sarcoma, ulceration can occur as a pressure necrosis over the summit. Probing under the ulcer edge is easily possible in these conditions but not in carcinomatous infiltration.



Fig. 16.8: Carcinoma breast with extensive skin involvement.



Fig. 16.9: Cancer en cuirasse with malignant nodules in chest wall and skin.

Swelling in the breast is an important finding to be inspected. Its location in relation to the quadrants of the breast, extent, size, shape, margin, surface, overlying skin should be examined.

Inspection of the axilla and supraclavicular fossa: Arm should be raised adequately to inspect the axilla. Axilla and supraclavicular fossa should be inspected for any lymph node swelling. Both sides should be inspected.

Inspection of arm and thorax: Oedema of the arm may be due to lymphatic obstruction of axillary nodes by malignant cells spreading from carcinoma breast. Oedema begins from distal to proximal and more

prominent distally (*brawny oedema*). Venous obstruction can also cause oedema arm. Here oedema is more prominent proximally in the arm and is having bluish discolouration over the skin. It is commonly due to infiltration and often by compression of lymph nodal metastatic disease onto the axillary vein. It needs urgent radiotherapy to axilla or chemotherapy otherwise venous gangrene of upper limb may develop. Arm oedema may be seen after mastectomy also (**Fig. 16.10**). Multiple nodules with skin thickening over the arm and chest wall due to carcinomatous infiltration is called as '*cancer en cuirasse*' as it looks like armor coat.

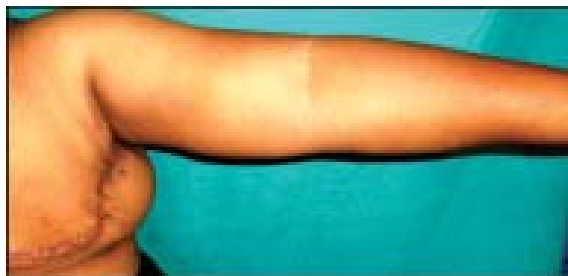


Fig. 16.10: Carcinoma of breast operated earlier with postoperative oedema arm. Note scar of mastectomy.

Palpation

Normal breast should be palpated first (**Fig. 16.12**). Palpation should be done using *the palmar aspect of the fingers with hand flat*. Normal breast tissue is firm, lobulated with fine nodularity. Often it can be soft and smooth also. Palpation is also done between thumb and fingers. All quadrants should be palpated along with nipple areola complex and axillary tail of Spence. During palpation one should look for raise in temperature over the breast (observed in mastitis but also can occur in vascular tumours like medullary carcinoma and sarcoma), tenderness, nature of the swelling—its size, shape, extent, surface, margin, consistency (carcinoma is hard/stony hard and irregular), fixity to breast tissue (swelling will not have independent/differential mobility), fixity to skin (by pinching the skin), fixity to pectoral fascia (by tethering), fixity to pectoralis major muscle/serratus anterior muscle/latissimus dorsi muscle. Palpate ulcer if present—look for tenderness, its edge and base for induration, bleeding on palpation. Nipple and areola should be palpated for tenderness, eversion, induration and discharge (**Figs 16.11A and B**).



Figs 16.11A and B: Palpation of breast lump with palmar surface of fingers. Palpation is also done between fingers and thumb.



Fig. 16.12: Normal breast should be palpated first. Then diseased side is palpated. It should be palpated for mass, its location, shape, size, surface, consistency, mobility and fixity.

Local rise of temperature: It is checked with dorsum of fingers. Breast is warm in mastitis and so also sarcomas can be warmer. Aggressive carcinoma also can be warm due to increased vascularity.

Tenderness: Breast is tender to palpate in acute mastitis and abscess. Carcinoma is nontender initially but becomes tender once skin is involved or when chest wall infiltration occurs.

Location of lump in the breast: Fibroadenoma is common in lower quadrant; fibroadenosis and carcinoma is more common in upper outer quadrant. One should remember that it is only incidence wise; in a given patient any disease can develop in any quadrant (**Figs 16.13 and 16.14A and B**).

Quadrants of breast

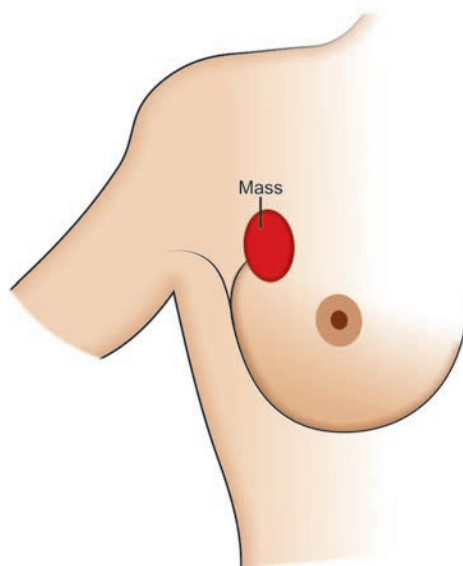
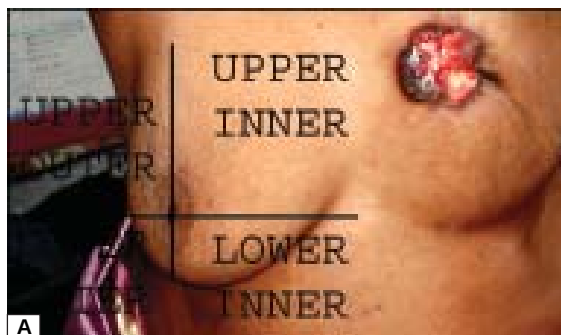
Quadrants of breast are marked by drawing two lines, vertical and horizontal along the nipple

- Upper outer quadrant (includes axillary tail also)—commonest site for carcinoma – 60%
- Lower outer quadrant – 10%
- Upper inner quadrant – 12%
- Lower inner quadrant—close to mediastinum – 6%
- Central quadrant—nipple and areola region – 12%



Fig. 16.13: Carcinoma breast over the commonest site—upper outer quadrant—more visible on raising the arm.

Number, size and shape: Carcinoma of breast is solitary; fibroadenosis can be multiple. Fibroadenoma is usually solitary but multiple fibroadenomas are known to occur occupying entire breast tissue. Opposite breast also can be involved especially in fibroadenosis. Size is important in staging the



B

Figs 16.14A and B: Quadrants of breast. Carcinoma is more common in upper outer quadrant as more breast tissue is located in this quadrant.

(T staging) carcinoma breast and so it should be measured using a tape (in cm). Fibroadenoma more than 5 cm is called as *giant fibroadenoma*.

Margin: Margin is well-defined and regular in fibroadenoma; well-defined and irregular in carcinoma; ill-defined in fibroadenosis.

Surface: It may be nodular or granular or uneven in carcinoma. Smooth surface is seen in benign condition like fibroadenoma.

Consistency: Fibroadenoma is firm swelling; carcinoma is stony hard; fibroadenosis is firm or diffuse India rubber consistency. Sarcoma is variable with soft or firm or hard in texture.

Fluctuation: When swelling is soft, fluctuation test is done. It is done by examiner standing or sitting behind the patient. Two hands of the examiner are placed above the shoulders of the patient. Swelling is held with one hand and with index finger of the other hand summit of the swelling is pressed/indented. Fluid displacement can be appreciated with yielding of the finger. Cystic swelling, localised abscess can be fluctuant. *Bloodgood cyst* is localised cystic swelling observed often in fibroadenosis of breast.

Transillumination test: It should be checked if there is clear fluid in the cyst. It is done using a torch ideally in a dark room.

Fixity of the lump to breast tissue: It is checked by holding the breast tissue in one hand and moving the lump in other hand. If lump is fixed to breast tissue, then breast tissue moves along the lump. Carcinoma breast is fixed to breast tissue. Fibroadenoma shows free mobility (differential mobility) within the breast tissue and so is called as 'breast mouse'.

Skin tethering can be demonstrated by moving the lump one side. It is due to inward puckering of the

skin following involvement of the elastic Cooper's ligament which becomes inelastic. Dimpling of skin appears which can be demonstrated by raising the arms above the shoulder level. When *skin tethering occurs lump can be moved in the arc anywhere without moving the overlying skin whereas lump cannot be moved at all without moving the skin in skin fixation (Fig. 16.15).*

Fixity to skin: When tumour directly infiltrates the skin, fixity occurs. Here skin will not be moved separately over the lump. Skin thickening and hard nodules are felt. *Peau d' orange* can be better seen by holding the skin between thumb and fingers. Whether benign or malignant, when tumour lies beneath the nipple, it is fixed to it. But tumour beneath the areola may or may not be fixed to it as it depends on presence or absence of infiltration to areola (**Figs 16.16A and B**).

Fixity to pectoralis major muscle: It is checked in sitting position. Patient is asked to keep her hands on her waist. Lump is moved along the direction of the muscle and also perpendicular to the direction of the muscle. Patient is asked to hold the hands tightly pressed over the waist to contract the pectoralis major muscle (action of the muscle is flexion of the shoulder) which is confirmed by feeling the taut muscle. Lump is again moved along the direction and perpendicular

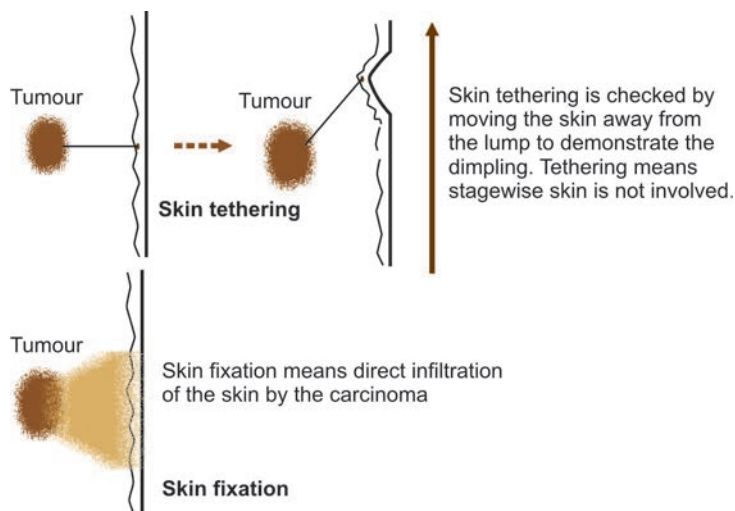


Fig. 16.15: Demonstration of skin tethering and fixation. Lump can be moved like an arc in skin tethering with demonstration of skin dimpling. Lump cannot be moved separately in skin fixation.



Figs 16.16A and B: Fixity to skin should be checked by pinching or holding the skin separately from tumour.

to the direction of the muscle. Mobility *along the line of muscle fibres* will be restricted totally if lump is adherent to the pectoralis major muscle. It becomes T₃ stage tumour (**Figs 16.17A to 16.18**).

Fixity to latissimus dorsi muscle: It is checked in sitting position with examiner standing by the side of the patient. Latissimus dorsi is an extensor of the shoulder joint. Initially mobility of the lump is checked and then arm is extended against resistance with elbow flexed 90° to contract the latissimus dorsi. If now mobility of the lump is restricted, it confirms that lump is fixed to latissimus dorsi muscle (**Fig. 16.19**).

Fixity to serratus anterior muscle: It is checked by checking the mobility of the lump before and after contracting the serratus anterior. Contraction of serratus anterior is achieved by pushing both the outstretched hands against resistance over the wall or over the examiner's shoulders and checking for restriction of mobility of the lump. It signifies involvement of chest wall—stage T₄ (**Figs 16.20A to C**).



Figs 16.17A to D: Checking for fixity to pectoralis major muscle. Muscle is made taut by keeping the patient's hands over the waist and mobility of lump are checked both in relaxed and contracted status of the muscle. Taut muscle should be confirmed by palpating the muscle in anterior axillary fold.



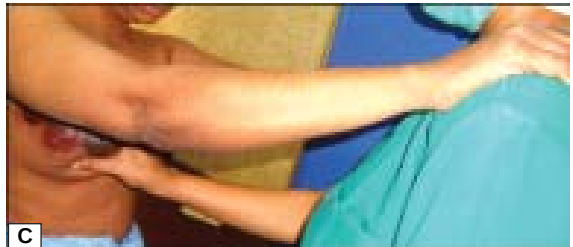
Fig. 16.18: Proper contraction and feeling of pectoralis major muscle is important.



Fig. 16.19: Fixity to latissimus dorsi muscle is checked by checking the mobility of the mass while extending the arm against resistance.

Chest wall fixity: It can be assessed by absence/presence of mobility of the mass; and breast with mass will not fall forward if it is fixed to underlying chest wall; and on raising the arm above shoulder breast with mass will not raise upward. Chest wall fixity means fixity to ribs and intercostals muscles (**Fig. 16.21**).

Palpation of nipple: It is equally important to palpate the nipple. Tenderness, thickening, hardness, mobility should be checked. Tumour underneath nipple is usually fixed to nipple. Retraction of nipple may be confirmed by palpating it. Discharge can be better appreciated while palpating the lump in the breast or other part of breast tissue or nipple itself. Colour, content (serous, blood, pus, greenish milk) of the discharge can be found. Discharge should be collected



Figs 16.20A to C; Fixity to serratus anterior is checked by checking the mobility of the lump while pushing both the outstretched hands of the patient, over the wall or over the examiner's shoulder against resistance.



Fig. 16.21: Fungating carcinoma breast. Note the extension of fungation into the chest wall.



Fig. 16.22: Typical retraction of the nipple.

for cytology or culture or AFB staining. In retracted nipple, gentle pressing of the base of the nipple is done to evert it. If it is due to congenital or of benign cause, retracted nipple can be everted by pressing at the base. If retraction is due to carcinoma, it cannot be everted at all. Retraction is *circumferential* in carcinoma; *slit like* in duct ectasia (**Fig. 16.22**).

Changes that can occur in nipple

- Destruction
- Depression (retraction)
- Discolouration
- Displacement
- Deviation
- Discharge
- Duplication

Palpation of areola: Areola should be palpated for nodularity, thickening, ulcer, destruction. Paget's disease can cause destruction of areola.

Examination of an ulcer over breast: Ulcer if present over the breast lump, should be examined like any ulcer with inspection of floor, margin, edge, discharge; palpation for tenderness, induration, mobility, fixity.

Examination of ipsilateral, regional axillary lymph nodes. Anterior/pectoral, central/medial, posterior, lateral, apical lymph nodes should be examined.

Supraclavicular lymph nodes should be examined.

Examination of opposite breast opposite axilla: Opposite axillary nodes are also examined. It may get involved through retrograde spread from internal mammary nodes or through cutaneous lymphatics (**Fig. 16.23**).



Fig. 16.23: Recurrent carcinoma breast with lesions in the scar, skin nodules, cancer en cuirasse, and peau d orange on the skin over opposite breast. It could be carcinoma of opposite breast probably as a spread from this side. De novo opposite side carcinoma is also possible.

Palpation of Axillary Lymph Nodes

It is an important step in examination of carcinoma breast (**Figs 16.24A to 16.26**).

Anterior/pectoral group of nodes are commonly involved nodes. Patient will be in sitting position. Raise the patient's arm high and inspect the axilla. Place the patient's forearm over examiner's forearm. Palpate the relaxed axilla over pectoralis major muscle for any lymph nodes. Examiner will use his left hand to examine the nodes (of right axilla) and his right hand will be over patient's left shoulder to support (**Fig. 16.27**).

Interpectoral nodes (Rotter's) are also palpated similarly by insinuating the fingers between the two pectori. It signifies retrograde spread of the tumour. It is often difficult to palpate.

Central/medial group of nodes are palpated in similar way like pectoral nodes but hand in the axilla is directed medially over the lateral chest wall and with gentle rolling movements using pulp of the finger (**Fig. 16.28**).

Lateral/humeral group of nodes are palpated with examiner's right hand (for right axilla) with left hand placed over *same side* shoulder (**Fig. 16.29**).

Posterior/subscapular nodes are palpated with patient in sitting position and examiner standing behind the



Figs 16.24A and B: Inspection of the axilla with raised arm is very important clinical method.



Fig. 16.25: Palpation of left axilla using right hand.



Fig. 16.26: Palpation of right axilla using left hand.



Fig. 16.27: Examination of pectoral group of lymph nodes.



Fig. 16.28: Examination of central group of lymph nodes.

patient. By raising the arm and forearm of the patient from opposite side the posterior axillary fold is palpated between thumb and fingers (**Figs 16.30A and B**).

Apical nodes are palpated (for right axilla) with left hand of the examiner placing high in the axilla with right hand supporting over the shoulder and supraclavicular region of the same side of the axilla. It is often difficult to palpate (**Fig. 16.31**).



Fig. 16.29: Examination of lateral group of lymph nodes.



Fig. 16.31: Examination of apical group of lymph nodes.



A



Fig. 16.32: Examination of supraclavicular group of lymph nodes.



B

Figs 16.30A and B: Examination of posterior group of lymph nodes.

Supraclavicular nodes are palpated using fingers over supraclavicular fossa by standing behind the patient who is asked to shrug the shoulder (**Fig. 16.32**).

Axillary nodes on opposite side are also examined. Opposite axilla can be examined by examiner standing on the same side by leaning over the patient or can be examined by standing on the opposite side. Its involvement signifies stage IV disease. It is confirmed by FNAC.

Levels of the axillary nodes (Berg's levels) (Fig. 16.33)

Level I—Below and lateral to the pectoralis minor muscle—anterior, lateral, posterior

Level II—Behind the pectoralis minor muscle—central

Level III—Above and medial to pectoralis minor muscle—apical

Note: Total number of nodes in the axilla is around 50.

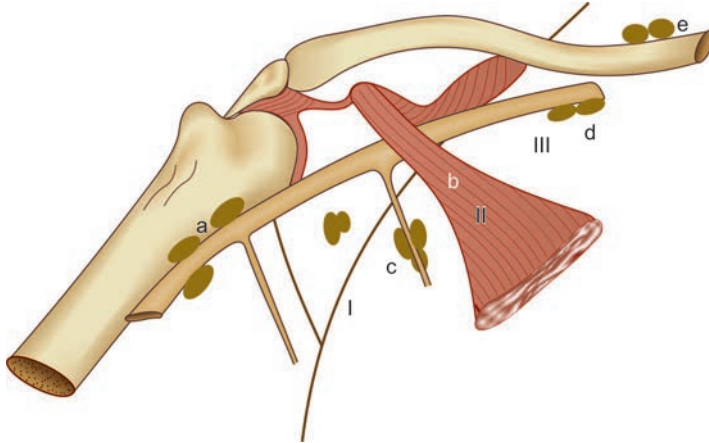


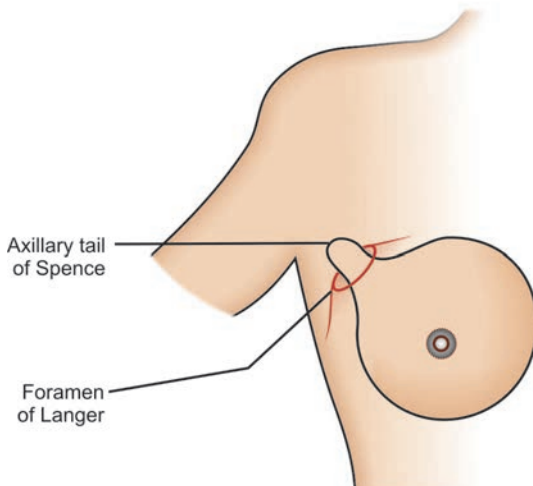
Fig. 16.33: Surgical levels of lymph nodes in the axilla draining from breast. a: Lateral group, b: Central group, c: Pectoral group, d: Apical group, e: Supraclavicular group.

Note

- Spread restricted to Level I nodes carries better prognosis
- Spread to Level II has poor prognosis
- Spread to Level III indicates worst prognosis

Axillary tail of the Spence: It is the extension of the upper outer quadrant of breast across foramen Langer deep to deep fascia. Foramen Langer is an opening in deep fascia over outer aspect of the breast which allows part of breast tissue to extend under deep fascia,

otherwise rest all breast tissue is in subcutaneous plane. Axillary tail is located adjacent to outer border of the pectoralis major muscle. When it is involved by carcinoma it should be differentiated by pectoral node enlargement. Axillary tail will move along with main breast tissue whereas pectoral node will not move when breast is moved as it has got independent mobility. Axillary tail often extends over the lateral edge of the pectoralis major muscle up to axilla (**Figs 16.34A and B**).



A



B

Figs 16.34A and B: Axillary tail of Spence. It is actually part of upper outer quadrant. Tumour in the axillary tail of Spence which is fungating.

Fixed enlarged axillary nodes can cause lymphoedema due to lymphatic block; venous thrombosis and venous oedema due to venous block; and severe excruciating pain along the distribution of the median and ulnar nerves (rare in radial nerve) with often significant sensory and motor deficits due to tumour infiltration of the cords of brachial plexus (usually medial cord occasionally lateral cord).

Examination of arms for venous oedema or lymphoedema: Venous oedema may be due to axillary vein compression by nodal mass. Lymphoedema may be due to lymphatic block following nodal involvement. Lymphoedema is mainly distal. It is gradual in onset and progressive. Venous oedema is sudden in onset, with bluish discolouration over the skin, uniform in both distal and proximal aspect of the upper limb (forearm and arm).

Examination for mediastinal node involvement: It is done by percussion. Initially percuss for liver dullness. Percussion is done one space above from lateral to medial, to look for widened mediastinal border. Mediastinal nodes are common in middle mediastinum.

Examination of respiratory system: It is done for secondaries—altered breath sounds, features of consolidation or pleural effusion are looked for (**Fig. 16.35**).



Fig. 16.35: Look for pleural effusion and altered breath sounds for secondaries in lungs.



Fig. 16.36: Always examine abdomen for liver enlargement, ascites or Krukenberg tumour (in premenopausal age).

Examination of abdomen: To look for palpable nodular liver, Krukenberg tumours in ovaries in menstruating age group, and ascites. It is completed with digital examination of rectum (P/R), and per vaginal examination (**Fig. 16.36**).

Examination of pelvis, spine, long bones for any swelling/tenderness/pathological fracture/restricted movements of spine, hips, etc.

Examination of central nervous system to look for any neurological deficits following metastatic disease in the brain.

Breast self examination (BSE) has got a major role in early detection of the carcinoma breast.

Ideally done once a month, just after the menstruation, as during this time breasts are less engorged. In postmenopausal age group it is done regularly at monthly intervals (fixed day of the month).

- Examine both breasts
- American cancer society recommends monthly BSE after 20 years of age
- Remind the patient that 90% of breast lumps are not cancer
- Better way is in lying down position with arm raised with a mattress support behind (**Fig. 16.37**)
- Palpation is done over all quadrants of the breast using the fingers
- If any doubtful swelling is palpable, consult the surgeon
- Nursing mother should perform BSE just after feeding the baby.

Assessment of nipple deviation: Nipple changes are assessed by inspection, palpation and measurement. Displacement of nipple is assessed by measuring

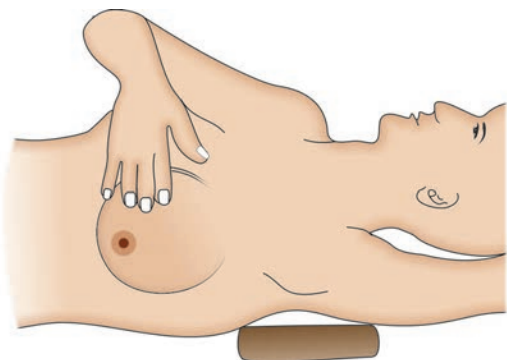


Fig. 16.37: Self-examination of breast is done in lying down position.

distance between mid-clavicular point to the nipple. This reveals any upward/downward displacement of nipple. Outward/inward displacement is assessed by measuring the distance of nipple from midline.

Cystic swellings of the breast

- Bloodgood cyst
- Breast abscess
- Hydatid cyst
- Galactocele
- Serocystic disease of Brodie
- Cystic necrosis in carcinoma breast
- Lymph cyst
- Haematoma in breast.

Causes of massive enlargement of breast

- Benign hypertrophy usually bilateral
- Giant fibroadenoma (> 5 cm)
- Serocystic disease of Brodie
- Sarcoma
- Carcinoma often when extensively involved
- Filariasis of breast.

Causes of hard swellings in the breast

- Carcinoma breast
- Antiboma breast
- Traumatic fat necrosis
- Calcified haematoma
- Fibroadenoma—hard variety.

Investigations in Carcinoma Breast/ Breast Lump

Mammography

It is plain X-ray of soft tissue of breast using low voltage and high amperage X-rays (300 MV and 40 KV). Two

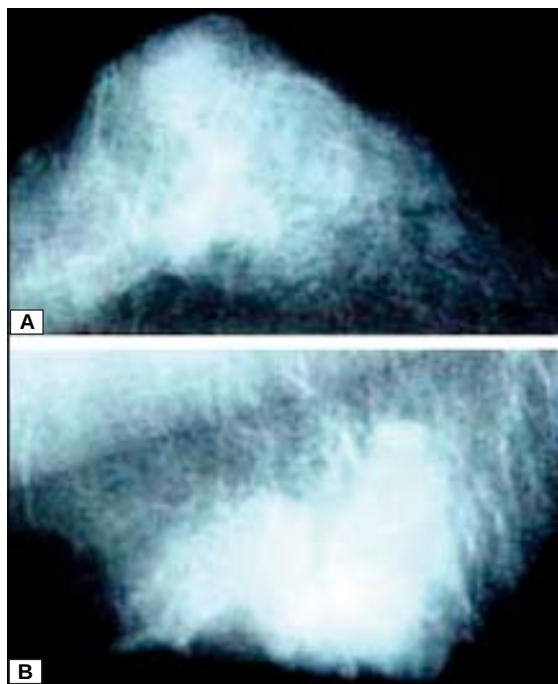


Fig. 16.38: Mediolateral and craniocaudal mammographic pictures showing microcalcification, spiculations and irregularity.

films are taken—*Craniocaudal* from above downward; *mediolateral* from side-to-side. Dose of radiation is 0.1 cGy, a low dose. So it is a safe and effective procedure (**Figs 16.38A and B**).

Findings

- Microcalcifications/branching calcification signify malignancy
- Soft tissue shadow may be smooth and regular in benign conditions or irregular in carcinomas
- Size and location of mass lesion is assessed with nipple and skin change (thickness)
- Spiculations; mammary duct distortion; loss of symmetry; clustering; architectural distortion suggests carcinoma.

Breast Imaging Reporting and Data System (BI-RADS) has got its own categories (0-5), assessment and recommendations. Digital mammography is computerised electronic image of the breast with enhanced magnified pictures. Digital spot-view mammography allows faster and more accurate stereotactic biopsy.

Indications for mammography: For screening purpose it is done after 40 years. Early screening is indicated

when there is family H/o carcinoma breast or histological risk factor. Mammography before 25 years of age is *usually not done* unless there is a lump or a strong family history or suspicious lump; in obese patients; to find out spread or de novo tumour in the opposite breast; mastalgias; mammography guided biopsy can be done; evaluation and follow up in benign breast disease with malignant potential; follow up mammography after conservative breast surgery. *Grading of lesions in mammography:* Grade I: Negative; Grade II: Benign lesion; Grade III: Probably benign lesion; Grade IV: Suspicious of malignancy; Grade V: Highly suggestive of carcinoma; Grade VI: Known carcinoma.

Digital mammography is better to analyse and store the pictures.

Xeromammography is same as above, but here a photoconductor is used to produce a final image on a Selenium paper (on aluminium plate) rather than on X-ray film. Such charged plate is placed behind the breast and X-ray exposure is done. Selenium positive charges are released, which depends on X-ray traversing the plate and the tissue. Electrostatic image of the breast produced is made blue by spraying negatively charged blue toner on the plate which attracts image of positive charges. This blue image of breast in a special coated plastic paper is fused permanently using heat. *Advantages:* Edge enhancement effect, therefore useful in dense breasts. *Disadvantage:* Exposure to high radiation dose and selenium plate is needed. Mammography of excised specimen may be useful in identifying the exact site of small lesion for histological assessment.

Contrast mammography often can be done by injecting water soluble iodine dye into the major lactiferous duct. Duct papilloma may show smooth filling defect; duct carcinoma shows irregular filling defect.

The condition when lump is clinically not palpable but mammogram shows identifiable carcinoma is ideal for breast conservative surgery like quadrantectomy/QUART therapy.

Ultrasound of Breast

It is done to look for lesion whether solid or cystic, margin of the lesion, internal echoes, retrotumour acoustic shadowing, compressibility, dimensions.

Irregular margin, irregular internal echoes, irregular posterior shadowing, non-compressibility, ratio between anteroposterior to width (lateral/horizontal) dimensions more than 1 are the features of carcinoma. Doppler will show high frequency signals with continuous flow. Benign lesions are smooth, rounded with well defined margins with weak internal echoes and compressibility. Disadvantage is lesions less than 1 cm may not be identified. FNAC can be done under US guidance. It is cheaper, easily available and there is no risk of radiation. It is preferred method of screening in pregnancy and early lactation (**Fig. 16.39**).



Fig. 16.39: US left breast showing fibroadenoma.

FNAC (Fine-Needle Aspiration Cytology)

FNAC is *very useful* in diagnosing the carcinoma breast. FNAC is also done under US guidance. But negative results are difficult to interpret because it may be due to sampling errors and so requires further diagnostic methods. FNAC of opposite breast, lymph nodes, opposite axillary lymph nodes are also often required. It is done with 23 gauge needle using FNAC aspiration special syringe. With the lump held firmly, the needle is passed into the lump and with negative pressure continuous aspiration is done until adequate material comes through the needle. Needle with syringe is removed without negative pressure. Material is collected on a slide; a smear is made using 100% alcohol. Cytology is studied after staining it under microscopy. After FNAC, cystic swelling often disappears completely. Excision may be indicated, if it recurs rapidly after two aspirations, and if FNAC is inconclusive (**Fig. 16.40**).



Fig. 16.40: FNAC of breast lump.

Advantages of FNAC: FNAC is least painful, can be done on OP basis, reliable and cheaper. Malignant deposits will not occur along FNAC track (only contraindication for FNAC is testicular tumour). **FNAC** is **Fine Needle Non-Aspirating Cytology**. **FNAC scoring:** C₀ – no epithelial cells; C₁ – scanty epithelial cells; C₂ – benign cells; C₃ – atypical cells; C₄ – suspicious cells; C₅ – malignant cells.

Reliability of FNAC and mammography

	FNAC	Mammography
Sensitivity (true positivity)	90-98%	90%
Specificity (without false positive)	98-100%	90%
False negative	2-10%	10%
False positive	Near 1-5%	10%

Frozen Section Biopsy

If FNAC fails even after two trials or in cases of negative FNAC, then *on table frozen section biopsy* is done for diagnosis. It has got 20% false negative results. It is often difficult to differentiate between severe atypia and carcinoma by frozen section and so its validity and use is under debate. In such situation *excision biopsy* is better. While doing excision biopsy incision is placed in such a way that it can be included in eventual mastectomy. **Trucut/core biopsy** is also used nowadays in many centres.

Chest X-ray: To look for pleural effusion, cannon ball secondaries in lungs, mediastinal lymph nodes, secondaries in rib. **CT chest** is *more reliable* method to see lung secondaries.

Ultrasound abdomen: To look for liver secondaries, ascites, and 'Krukenberg' tumour.

X-ray spine or MRI spine/pelvis shows osteolytic secondaries in the bone like vertebra and pelvic bones.

Radioisotope bone scan is done to look for secondaries in bone in advanced cases. It is not done routinely

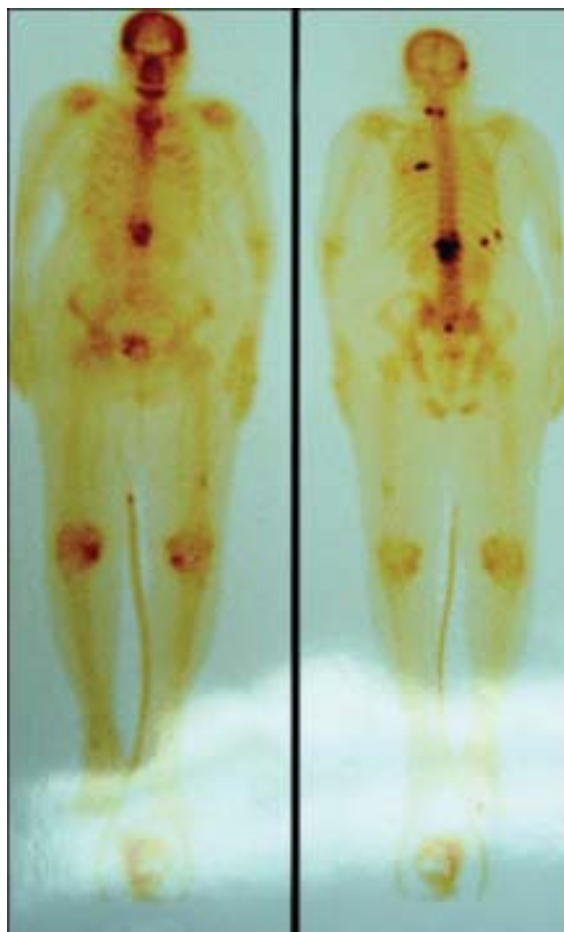


Fig. 16.41: Radioisotope bone scan is done to see bone metastasis.

in early carcinoma of breast. Healed fractures; Paget's bone disease, osteoarthritis may show hot spots – accounting for false positive pictures. Bone biopsy in suspected area may be confirmative even though it is not advocated commonly (**Fig. 16.41**).

Indications for whole body bone scan in carcinoma breast

- T₃, T₄ advanced disease
- Advanced nodal disease
- Bone pain, bone swelling, pathological fracture
- Chest/liver secondaries

Oestrogen Receptor Study

They are oestrogen sensitive receptors, which are cytosolic glycoprotein present in the breast and tumour

tissue. It is an important indicator of prognosis of carcinoma breast. Tissue for receptor study is sent in low temperature in ice flasks. It is assessed by quantitative analysis (Frozen -70°). If value is more than 10 units (Femtomoles) per ng gram tissue it is called as *ER +ve status*. If value is 5-9 it is borderline and if it is less than 5 femtomoles per nanogram tissue it is called as *ER -ve status*. **In ER +ve status**—Prognosis is good; Hormone therapy including Tamoxifen is very beneficial; Response to treatment is better. **In ER -ve status**—Prognosis is poor; Hormone therapy is not very beneficial (but used) as compared to ER +ve patients; Response to treatment is not good. ER positivity is common in postmenopausal women (60%) compared to premenopausal women (30%). *Progesterone receptor (PR status)* study and Her 2 Neu receptor status are other studies done at present to plan the therapy and assess the prognosis.

Study of Discharge from the Nipple

Nipple discharge is usually unilateral in carcinoma breast. **Ductal lavage** may be useful in some patients. Microcatheter of 1 cm length is introduced gently into the ductal opening. 10 ml saline is infused through the catheter. Fluid is withdrawn into the syringe and cytological analysis is done.

MRI of Breast and MRI of Spine (in case of suspected spine secondaries)

It is done—to differentiate scar from recurrence; to image breasts of women with implants; to evaluate the axilla and recurrent disease. Both precontrast and postcontrast MRI are done. T_1 and T_2 -weighed images are taken. Irregular mass with spiculations, changes in skin and nipple, lymphoedema are the findings in carcinoma breast (**Fig. 16.42**).

Edge Biopsy

Edge biopsy is done only when there is skin involvement—ulceration and fungation. Diathermy should be avoided in incision biopsy as it may distort the histology of tumour and study of hormone receptor status may not be possible.

Tumour Markers

They are used mainly during follow up period. CA 15/3 is commonly done when needed.



Fig. 16.42: MRI spine showing secondaries primary being carcinoma breast.

Sentinel Lymph Node Biopsy (SLNB)

The first axillary (SLN) node draining the breast (by direct drainage) is designated as the *sentinel node*. SLN is first node involved by tumour cells and presence or absence of its histological involvement, when assessed will give a predictive idea about the further spread of tumour to other nodes. Involvement of other nodes without SLN is less than 3% and so if SLNB is negative nodal dissection can be avoided but regular follow up is needed. SLNB is done in all cases of early breast cancers, T_1 and T_2 **without clinically palpable node**. It is not done in clinically palpable axillary node as there is already distortion of lymphatic flow due to tumour. It is also not done in multifocal and multicentric tumours, as there is involvement of many lymphatic trunks from different quadrants of breast, chances of false negative is high. Sentinel node is localised by preoperative (within 12 hours) or per-operative injection of patent blue (Isosulfan vital blue dye) or ^{99m}Tc radioisotope labeled colloid albumin near the tumour (peritumour area). Marker will pass through the sentinel node which can be detected

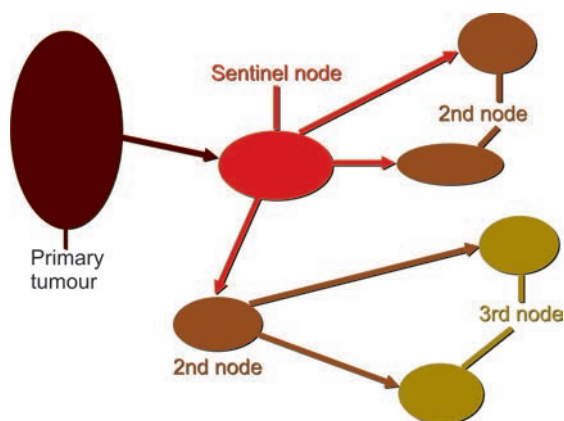


Fig. 16.43: Sentinel Lymph Node Biopsy (SLNB) of breast. Note: Spread by skipping the sentinel node is less than 3%.

visually as blue staining or with a hand held gamma camera; and is biopsied with a small incision directly over it. If there is no involvement of sentinel node by tumour then further axillary dissection is not required as *skip lesions* (skipping sentinel node) occur only in less than 3% cases. Note: Facility for SLNB is not available in many centres (**Fig. 16.43**).

SLNB is done in—

- Carcinoma breast
- Carcinoma penis
- Malignant melanoma.

Axillary sampling is often done with an adequate axillary incision. 10-15 nodes are removed for sampling. It is not commonly practiced now (Minimum 10 nodes should be removed—level I nodes).

CT scan of chest, abdomen and brain whenever needed. CT is said to be more useful to detect secondaries in these regions.

Triple assessment—It includes (1) Clinical assessment; (2) Radiological imaging (US in young, mammography in adult); (3) Cytological (FNAC) or histological analysis (core biopsy).

Ductography

It is contrast study of ducts of breast in case of unilateral nipple discharge. Fine cannula is passed under vision carefully through the duct opening into the duct and 0.2 ml of dilute water-soluble contrast media is injected

into the duct. Craniocaudal and mediolateral X-ray films are taken. Contrast irregular filling defect may be observed.

Thermography is not very sensitive test (50%). Malignant tumours are hypervascular and so transmitted temperature is detected through different thermographic methods. It is the pictorial representation of infrared emission of breast.

Biochemical analysis: Increased serum alkaline phosphatase, γ glutamyl transaminase suggests liver secondaries. Raise in urinary hydroxyproline means collagen breakdown suggesting secondaries. A low value of urinary aetiocholanolone, a metabolite of adrenal dehydroepiandrosterone in relation to total 17 hydroxycorticosteroids (in urine) is specific of breast carcinoma suggesting poor prognosis and poor response to adrenalectomy and hypophysectomy (Poor discriminants).

Newer modalities of investigations: Stereotactic core biopsy using computer mammography; vacuum assisted biopsy using 11 gauge biopsy probe; needle localised biopsy under mammographic guidance; I^{125} localisation biopsy.

Differential Diagnosis

Carcinoma Breast

How carcinoma breast is suspected?

Any lump in the breast can be malignant unless proved otherwise. But one has to remember that every breast lump need not be always malignant. Short duration, rapid progression, nodal status, hard consistency, with often irregular surface and late features like fixity to chest wall, ulceration/fungation and distant spread are the features to consider for carcinoma breast. *Painless lump with irregular surface and stony hard consistency* are typical features of carcinoma breast.

One has to remember that differential diagnosis for carcinoma breast is same as all conditions which are benign diseases of the breast (**Table 16.1**).

Table 16.1: Differential diagnosis for carcinoma breast/benign diseases of breast

• Fibroadenosis	• Mastitis
• Traumatic fat necrosis	• Antibiooma
• Tuberculosis of breast	• Galactocele
• Bloodgood cyst	• Mondor's disease
• Filariasis breast	• Cystosarcoma phylloides

Aetiology: Carcinoma breast is more common in developed western countries. In African-American women, it is more aggressive. It is less common in Japan. It is second most common carcinoma in females. Incidence is 19-34%. Median age is 47 years. It is more common after middle age, but can occur at any age group after 20. It is familial in 2-5% cases. Carcinoma in one breast increases the risk of developing carcinoma on opposite breast by 3-4 times. Incidence of bilateral carcinoma is 2%. Diet low with phytoestrogens and high alcohol intake may predispose carcinoma. It is common in nulliparous women. Early child bearing and breastfeeding reduces the incidence of malignancy. Breast carcinoma is directly related to oestrogen level increase. Early menarche and late menopause has got higher risk probably due to increased oestrogen level. It is more common in obese patient. Breast cancer relative risk is qualified as Relative Risk (RR). RR 2.0 means risk is twice the normal population. If RR is 0.5 means risk is 50% less than normal population. In males, occasionally gynaecomastia turns into carcinoma. Benign breast diseases with atypia, hyperplasia and epitheliosis have got higher risk in a patient with family history of carcinoma breast; mutation of tumour suppressor genes BRCA 1 and BRCA 2 has also shown high risk of carcinoma breast. BRCA 1 has got more risk (35-45%). It is located in long arm of chromosome 17. It is also associated with ovarian carcinoma. It is poorly differentiated, ductal invasive, hormone receptor negative type carcinoma BRCA2 is located in long arm of chromosome 13. It is well-differentiated, invasive, positive hormone receptor type carcinoma. It is also associated with carcinoma male breast. Occasionally mutation of BRCA3 and p53 suppressor gene is also involved. Cowden's syndrome and Li-Fraumeni's syndrome are associated with carcinoma breast. Presently carcinoma breast is considered as systemic disease. Her 2 neu mutation (transmembrane growth factor) is seen in 80% invasive ductal carcinoma;

carries poor prognosis. *Halsted concept* of spread is *sequential spread*. Breast—axillary lymph node—systemic spread. *Fischer concept* is early to begin with itself, there is possibility of distant blood spread because of *micro metastasis* without nodal disease. Only tumour lesser than 1 cm size can be sequential. *Spectrum concept* is new one where disease spreads locoregionally as well as systemically which makes it to have both locoregional as well as systemic disease control. Prior diagnosis of uterine/ovarian/colonic cancers increases the risk of breast cancer. Endocrine cause is considered as '*Koreman's hypothesis*'. It means, anovulatory cycle which is more common in early menarche and late menopause with unopposed estrogen without progesterone; hyperprolactinaemia due to late child birth (which is inhibited in early child birth); oral contraceptives; absent breastfeeding; hormone replacement therapy; all predisposes carcinoma breast. Carcinoma breast shows geographical variation with very less incidence in Japan.

Incidences in carcinoma breast

- 30% of all female cancers
- 20% of cancer related deaths in females
- 2-4% bilateral
- 2-5% hereditary
- Familial breast cancer – 25%
- Sporadic breast cancer 70%
- Lump in the breast—commonest presentation (75%)
- 10% presents with pain
- 35-45% with mutation of BRCA1 gene
- 70% blood spread occurs to bones

Classification of Carcinoma Breast

Classifications

- I. Ductal carcinoma – 90%
Lobular carcinoma – 10%
- II. a. *In situ* carcinoma – no invasion through basement membrane.
 - DCIS (Ductal carcinoma in situ) – 5-20% - papillary, cribriform, solid, comedo types.
 - LCIS (Lobular carcinoma in situ) – 2%.

Li-Fraumeni syndrome	Cowden's syndrome	Ataxia telangiectasia
• P53 gene mutation • Autosomal dominant • Breast cancer • Osteosarcoma, leukaemia, soft tissue sarcoma, adrenocortical tumours	• Autosomal dominant • Multiple hamartoma syndrome • Facial trichilemmoma • Papilloma, GI polyps, thyroid tumours • Breast cancer	• Haemangioma • Breast cancer

Van Nuy's prognostic index for DCIS

Score	1	2	3
Size in mm	< 15 mm	15-40 mm	> 40 mm
Clearance in mm	> 10 mm	1-10 mm	< 1 mm
Grade and necrosis	Not high grade No necrosis	Not high grade Necrosis present	High grade Necrosis present
Total score is 9			
Score 3-4	Conservative breast surgery (wide local excision)		
Score 5-7	Conservative surgery + Radiotherapy		
Score 8-9	Total mastectomy		

Nottingham Prognostic Index (NPI) – $(0.2 \times \text{Tumour size in cm}) + \text{Lymph node stage} + \text{Tumour grade}$ **For nodes—1** – nodes; **2** is 1 to 3 nodes; **3** is 4 or more nodes**For grading 1,2,3**

NPI score	< 3.4	Good prognosis with 80% survival (15 years)
NPI score	3.4-5.4	Moderate prognosis with 40% survival
NPI score	> 5.4	Poor prognosis with 15% survival

b. Invasive—invasion through basement membrane—80%.

- Invasive ductal carcinoma—70%.
- Invasive lobular carcinoma. It is commonly multifocal and often bilateral. It is 10-15%.

III. Unilateral.

Bilateral: 2-5% common.

IV. Unifocal.

Multifocal.

Multifocal—tumour tissues within the same quadrant.

Multicentric—tumour tissues within the breast but in different quadrant.

Duct carcinoma in situ (DCIS): It is intraductal carcinoma without any invasion into the basement membrane. It is 5-20% common. It can be—*Solid*; *Comedo* with necrosis is high grade with increased chances of microinvasion; *Cribriiform*; *Papillary*, *Micropapillary*. It is associated with high expression of C – erb2 gene (80%). Nipple discharge and often-small swelling are main presentations. US assisted FNAC and mammography are the needed investigations.

Risk of lymph node spread in DCIS is less than 4%. So axillary dissection is not necessary.

Sentinel Lymph Node Biopsy and later required surgical procedure is the preferred method (if facility is available).

Pathological Types

Scirrhus carcinoma: It is 60% common. It is hard, whitish, or whitish yellow, noncapsulated, irregular, with gritty, *cartilaginous* consistency. It contains malignant cells with fibrous stroma.

Medullary carcinoma: It is also called as 'encephaloid type' because of its brain-like consistency. It contains malignant cells with dispersed lymphocytes. It is 4-10% common; occurs in younger age group; associated with BRCA 1 hereditary cancers; it is soft and often haemorrhagic; it carries better prognosis than scirrhus type.

Inflammatory carcinoma/lactating carcinoma/Mastitis carcinomatosis: It is 2% common; it is common in lactating women or pregnancy; it mimics acute mastitis because of its short duration, pain, warmth and tenderness. Clinically, it is rapidly progressive tumour of short duration, often involving whole of breast tissue with occurrence of *Peau d'orange*, often extending to the skin of chest wall also. It rapidly metastasizes to chest wall, bone and lungs. **It is always stage IV carcinoma.** FNAC confirms the diagnosis—It contains undifferentiated cells. It mimics acute mastitis but total count is normal in inflammatory carcinoma of breast.

Differences between Paget's disease and eczema of nipple		
	Paget's Disease (Jame's Paget-1874)	Eczema
1	Unilateral	Bilateral
2	Edges are distinct	Edges are indistinct
3	Itching absent	Itching present
4	Seen in menopausal women / elderly	Occurs during the time of lactation
5	Vesicles absent	Vesicles present
6	Nipple is usually destroyed	Nipple is usually intact
7	Underlying lump is usually present	No underlying lump

Colloid carcinoma (2%): It produces abundant mucin; common in elderly; presents as bulky tumour; positive for oestrogen receptors.

Paget's disease of the nipple: It is superficial manifestation of an intraductal carcinoma. The malignancy spreads within the duct up to the skin of the nipple and down into the substance of the breast. It mimics eczema of nipple and areola. In Paget's disease, there will be a hard nodule just underneath the areola, which later ulcerates and also causes destruction of nipple. Histologically it contains large, ovoid, clear Paget's cells with malignant features (**Figs 16.44 and 16.45**).

Tubular (2%), Papillary (2%), Cribriform are the other types of duct carcinomas. Papillary carcinoma occurs



Fig. 16.44: Paget's disease of the breast.



Fig. 16.45: Subareolar carcinoma with destruction of nipple-areolar complex.

in late age group – 7th decade; small tumour of less than 3 cm; carries good prognosis. Tubular also carries very good prognosis.

Atrophic scirrhous carcinoma: It is seen in elderly females. It is a slow growing tumour which has got better prognosis.

Lobular carcinoma in situ: It is predominantly premenopausal; need not be detected by mammography; it is an incidental pathological entity; multifocal and bilateral; clinically, it does not form a lump; does predispose to invasive cancer; 50% cancers can develop in the contralateral breast; immunohistochemistry using cadherin antibody shows positive reaction; it carries poor prognosis.

Disease of Reclus: It is rare intracystic papilliferous carcinoma of breast presenting as a cystic swelling with bloody discharge from the nipple.

Presently invasive breast cancer is classified as ductal and lobular. 70% are invasive breast cancers. It can be infiltrating ductal carcinoma not otherwise specified (NOS); tubular (2%); colloid (2%); medullary (basal like, triple negative means ER/PR/Her 2 neu negative, aggressive, 5%).

Spread of carcinoma breast: To lymph nodes by lymphatic permeation upto axillary nodes; through lymphatic embolization from axillary nodes further. Opposite breast and axillary nodes may be involved through internal mammary nodes and dermal lymphatics. Spread through blood can occur to bone (lumbar vertebra, pelvis, and femur), liver, lungs, and brain.

Causes of lymphatic block in carcinoma breast

- Involvement and fixation of the axillary nodes level I, II and III
- After levels I, II and III dissection
- After radiotherapy to axilla
- Inoperable fixed nodes in axilla
- Recurrent axillary disease
- May be associated with cancer en cuirasse
- Secondary infection

Effects of lymphatic obstruction

- Peau d' orange
- Brawny oedema of arm – indurated, painful, nonpitting – occurs in fixed nodes in axilla
- Elephantiasis chirurgens – after radical mastectomy or radiotherapy to axilla
- Cancer en cuirasse – seen in locally advanced carcinoma of breast. Skin of chest wall is studded with hard fixed nodules like armour coat (of soldiers)
- Lymphangiosarcoma after radical mastectomy or MRM (Stewart-Treves's)

Clinical features: A palpable lump which is painless initially, irregular surface, stony hard in consistency, *fixity to breast tissue* - are typical of carcinoma breast. Nipple retraction, dimpling, puckering of skin, tethering on palpation, skin fixation, ulceration, fungation, fixity to pectoralis major, latissimus dorsi and serratus anterior muscle; fixity to chest wall; palpable axillary nodes which are mobile initially but later gets fixed; palpable supraclavicular lymph nodes; palpable opposite axillary node; blood spread to bone, lungs, liver and brain—are different clinical features (**Figs 16.46 to 16.48**).

Staging of Carcinoma Breast

Manchester Staging:

1. Tumour in the breast, not involving pectoral or deeper plane. Skin involvement if present, it is lesser than the size of tumour. Lymph nodes are not palpable.
2. Same as stage I but with *mobile, discrete lymph nodes* palpable in the ipsilateral axilla.



Fig. 16.46: Advanced carcinoma left breast involving entire breast with nodularity.



Fig. 16.47: Recurrent carcinoma of breast with fungation. Note the scar of previous surgery.

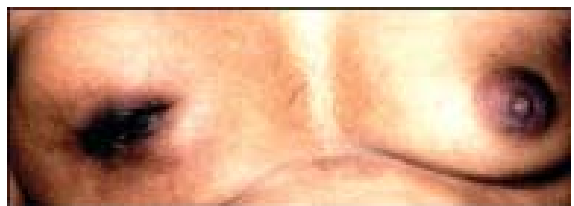


Fig. 16.48: Carcinoma right breast with retraction of nipple and areolar changes.

3. Tumour fixed to pectoral muscle or skin involvement more than the tumour size or ipsilateral axillary lymph nodes adherent to each other.
4. Tumour fixed to the chest wall, 'cancer en cuirasse', skin involvement wider than that of the breast or ipsilateral or contralateral side supraclavicular lymph nodes or opposite breast or opposite axillary lymph nodes or spread to bone, lung, liver or inflammatory carcinoma of breast.

TNM Staging (AJCC Cancer staging manual 2002 sixth edition):

Tumour:

- T0 No evidence of primary
 Tis Carcinoma in situ (DCIS or LCIS)
 Tis Paget's – Paget's disease of nipple with no tumour (with tumour underneath is staged according to size)
 T₁ Tumour size <2 cm in greatest diameter (T_{1a}-0.1-0.5 cm, T_{1b}-0.5-1.0 cm, T_{1c}-1-2 cm)
 T₂ Size 2-5 cm.
 T₃ Size >5 cm
 T₄ Tumour fixed to chest wall or skin (T_{4a}-fixed to chest wall, T_{4b}-fixed to skin, T_{4c}-T_{4a}+T_{4b}, T_{4d}-inflammatory carcinoma breast).

Node:

- N₀ No nodes.
 N₁ Axillary nodes – ipsilateral, mobile, discrete.
 N₂ –
 N_{2a} Axillary nodes – ipsilateral fixed to one another and other structures.
 N_{2b} Clinically apparent ipsilateral internal mammary nodes in the absence of clinically palpable axillary nodes.
 N₃ –
 N_{3a} Spread to ipsilateral infraclavicular lymph nodes with or without axillary nodes.
 N_{3b} Spread to ipsilateral internal mammary nodes and axillary nodes.
 N_{3c} Spread to ipsilateral supraclavicular lymph nodes with or without axillary or internal mammary nodes.

Metastasis:

- M₀ No metastasis.
 M₁ Distant metastases.

Stage I:	T ₁ N ₀ M ₀
Stage IIa:	T ₀ N ₁ M ₀ ; T ₁ N ₁ M ₀ ; T ₂ N ₀ M ₀ .
Stage IIb:	T ₂ N ₁ M ₀ ; T ₃ N ₀ M ₀
Stage IIIa:	T ₀ N ₂ M ₀ ; T ₁ N ₂ M ₀ ; T ₂ N ₂ M ₀ ; T ₃ N ₁ M ₀ ; T ₃ N ₂ M ₀
Stage IIIb:	T ₄ N ₀ M ₀ ; T ₄ N ₁ M ₀ ; T ₄ N ₂ M ₀
Stage IIIc:	Any TN ₃ M ₀
Stage IV:	Any T, any N, M

Early invasive breast cancer – Stage I, IIa, IIb. Locally advanced breast cancer – Stage IIIa/b/c. Distant spread – Stage IV.

The Columbia Classification (Haagsen, Cooley, and Stout)

Grave signs:	
Oedema of skin	Stage A—Only tumour. No Grave signs
Skin ulceration	Stage B—tumour + axillary lymph nodes < 2.5 cm
Fixity to chest wall	Stage C—tumour + any one of five Grave signs
Axillary lymph nodes >2.5 cm	Stage D—two or more Grave signs
Fixed axillary lymph nodes	Supraclavicular lymph node involvement Distant metastasis

Stage A—No skin oedema, ulceration, or fixation to chest wall. Axillary nodes are not clinically involved.

Stage B—Clinically involved axillary nodes less than 2.5 cm in diameter; Not fixed.

Stage C—Grave signs of comparatively advanced carcinoma; oedema of skin, skin ulceration, fixation to chest wall; Massive axillary involvement with nodes >2.5 cm in diameter; Axillary fixation.

Stage D—Advanced carcinoma including two or more signs in stage C; in addition satellite nodules; supraclavicular nodes; inflammatory cancer, arm oedema or distant metastasis.

Locally advanced carcinoma of breast (LACB): It means locally advanced tumour with muscle/chest wall involvement, extensive skin involvement or fixed axillary nodes. It will be T₃, T_{4a}, T_{4b}, T_{4c} or T_{4d} or N₂ or N₃. It is investigated by FNAC of tumour, mammography of opposite breast, chest CT, CT

abdomen or whole body bone scan. Treatment of LACB is always palliative by simple mastectomy, chemotherapy and hormone therapy using tamoxifen. Palliation is to control pain, to prevent fungation or bleeding. 5 year survival is 40% and 10 year survival is less than 25%. Inflammatory carcinoma is T_{4d} LACB. It is also called as *mastitis carcinomatosa* or *lactating carcinoma of breast*. It is 2% common. It is observed in younger age group usually in pregnancy or lactating period. There will be extensive skin involvement with pain. It often mimics mastitis of lactation. FNAC or incision biopsy concludes diagnosis. It is treated by initial chemotherapy or radiotherapy; later if tumour reduces in size then total mastectomy with axillary clearance can be done. But most often it is inoperable. After surgery, chemotherapy and tamoxifen is given. 5 year survival for inflammatory carcinoma of breast is 25-30%.

Metastatic carcinoma of breast: It is blood spread into different places like bone, lungs and pleura, liver, soft tissues, brain and adrenals. It is evaluated by FNAC/incision biopsy, chest CT, LFT, US abdomen, CT abdomen, whole body bone scanning, CT brain, tissue study for ER /PR/ HER-2 neu receptor status.

Common sites of distant spread in carcinoma breast

- Bones—70% —(lumbar vertebrae, pelvic bones, long bones)
- Lungs and pleura—20-30%
- Soft tissues—5-15%
- Liver—10-12%
- Brain—2-5%
- Adrenals—2-5%

Bone secondaries in carcinoma breast (Fig. 16.49)

- Commonest site of blood spread (70%)
- Common in lumbar vertebrae, femur, pelvis
- Pathological fracture can occur
- Can present with spinal compression and paraplegia
- Radiotherapy, internal fixation, spinal decompression is required
- Biphosphonates 1600 mg/day is beneficial

Malignant pleural effusion as secondaries from carcinoma breast (Fig. 16.49)

- It signifies terminal event
 - It has got poor prognosis
 - HRCT is ideal diagnostic tool.
 - Respiratory distress and failure is the main feature
- Treated by:* Intercostal tube drainage; pleurodesis using talc/ tetracycline; Chemotherapy



Fig. 16.49: X-ray showing typical osteolytic bone secondaries in pelvis in carcinoma breast.

Carcinoma breast in pregnancy

- Incidence is 3%
- Treatment is modified radical mastectomy (MRM)
- Chemotherapy can be given in 2nd trimester with care; Radiotherapy has no role
- As commonly ER negative, hormone therapy is not used. When distressing secondaries are present termination of pregnancy may be required
- *Women with breast cancer can become pregnant 2 years after the completion of therapy, as recurrence is more common within 2 years.*

Carcinoma of male breast (Fig. 16.50)

- It is less than 1% of cases of breast cancers. Commonly associated with BRCA 2 gene mutation
- Gynaecomastia and excess oestrogen are said to be the aetiological factors
- Commonly it is *infiltrating duct carcinoma*. Commonly ER positive (80%)
- Presentation, spread, behaviour are same as carcinoma of female breast. Investigations and treatment are same as carcinoma female breast. It is more aggressive than in females
- *Tamoxifen* is very useful in carcinoma male breast. *LHRH agonists* are next option. Earlier bilateral orchidectomy was the preferred choice. Now not commonly done.

Complications after mastectomy with axillary clearance are lymphoedema and eventual lymphangio-



Fig. 16.50: Carcinoma male breast showing mass, puckering and retraction.

sarcoma (after 3 to many years later) of the limb (*Stewart-Treves syndrome*). After chemotherapy alopecia is seen (**Fig. 16.51**).

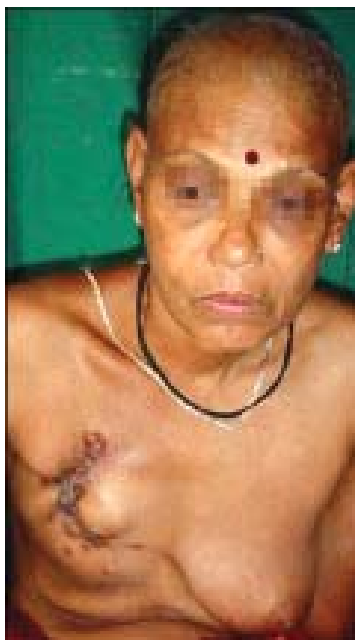


Fig. 16.51: Alopecia after chemotherapy to carcinoma breast.

Fibroadenoma

It is a benign encapsulated tumour occurring commonly in young females of 15-25 years age group. Presently it is considered as hyperplasia of a single lobule of the breast (may be classified under ANDI).

Types: Gross: Soft; Hard; Giant (> 5 cm in size).
Microscopy: *Pericanalicular*—small and hard—mainly fibrous; *Intracanalicular*—large and soft—mainly cellular.

Clinical features: It presents as a painless swelling in one of the quadrants, which is *smooth, firm, non-tender, well localised and moves freely* within the breast tissue—‘*mouse in the breast*’. It is common in lower quadrant but can occur in any quadrant. Axillary lymph nodes are not enlarged (**Fig. 16.52**).

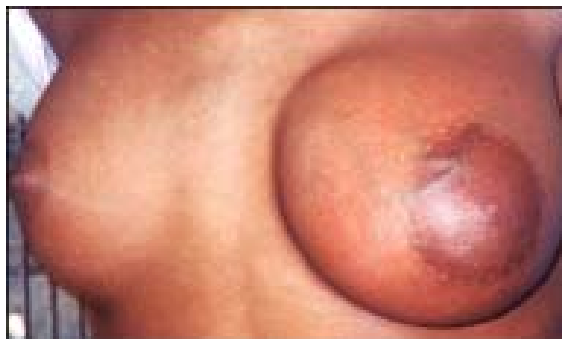


Fig. 16.52: Fibroadenoma left breast.

Investigations: Mammography (well localised smooth regular shadow); FNAC; Ultrasound (to confirm solid nature).

Points to be remembered:

- Size of giant duodenal ulcer is > 2 cm
- Size of giant gastric ulcer is > 3 cm
- Size of giant fibroadenoma is > 5 cm
- Diameter of transverse colon in toxic megacolon is > 6 cm
- Giant naevus is > 20 cm

Fibrocystadenosis (*Fibrocystic Disease of the Breast/Mammary Dysplasia*)

It is due to **Aberration of Normal Development and Involution (ANDI)** of breast causing—Fibrosis; Cyst formation; Glandular proliferation (Adenosis); Hyperplasia (Epitheliosis); Papillomatosis. It is an oestrogen dependent condition. One of the cysts may get enlarged

to become a clinically palpable well localized swelling - *bluedome cyst of Bloodgood*. Diffuse, small, multiple cysts in fibrocystadenosis is called as *Schimmelbusch's disease*. Disease is common in upper outer quadrant.

Clinical features: Presentation is during menstruating age group as a *bilateral, painful, diffuse, granular, tender, swelling* which is better felt with palpating fingers (poorly felt with palm). It is not fixed to skin, muscle or chest wall. Pain and tenderness are more during menstruation (*Cyclical mastalgia*). It subsides during pregnancy, lactation and after menopause. Discharge from the nipple when present will be serous or greenish. Greenish discharge is due to mixture of serous exudates and ductal epithelial cell debris.

Investigations: FNAC (Epitheliosis, when florid is undoubtedly premalignant); Ultrasound, mammography.

Aberration of Normal Development and Involution (ANDI) of the Breast

ANDI includes variety of benign breast disorders occurring at different periods of reproductive life in females—early, matured and involution phase of reproductive age group. It was first coined at Cardiff Breast Clinic in 1987 by *LE Hughes*. All conditions under ANDI should be carefully clinically examined and often mammography and FNAC/core cut biopsy should be done to rule out malignancy. ANDI includes different aberrations and diseases.

In early reproductive age group (15-25 years): Normal lobule formation may cause aberration as *fibroadenoma*. If it is more than 5 cm it is called as giant fibroadenoma as a diseased status. It is AND of a lobule. Normal stroma may develop juvenile hypertrophy as aberration and multiple fibroadenoma as diseased status.

In mature reproductive age group (25-40 years): Normal cyclical hormonal effects on glands and stroma get exaggerated by aberration causing generalised enlargement. Its diseased status is cyclical mastalgia with nodularity termed as *fibrocystadenosis*.

Involution age group (40-55 years): Lobular involution with microcysts, fibrosis, adenosis, apocrine metaplasia and eventual aberrations as macrocysts and

cystic disease of breast. Macrocyst is an aberration of normal involution (ANI). Sclerosing adenosis is also a type of aberration. *Ductal involution* may cause ductal dilatation and nipple discharge as aberration. Later disease status develops with periductal mastitis, bacterial infection, nonlactational breast abscess and mammary duct fistula. Periductal fibrosis may cause partial nipple retraction. *Epithelial changes* leads into epithelial hyperplasia and atypia.

Phylloides Tumour (Cystosarcoma Phylloides/ Serocystic Disease of Brodie)

They are not simply giant fibroadenoma. They show a wide spectrum of activity, varying from almost a benign condition to a locally aggressive and sometimes with a metastatic potentiality to lungs through blood. *Benign phylloides* is commonest type which needs excision; *borderline phylloides* is intermediate in nature which needs wide local excision with 1 cm margin; *malignant phylloides* is rare but an aggressive sarcoma spreading through blood commonly to lungs and needs total mastectomy without axillary nodal addressing but carries poor prognosis. Depending on mitotic index and degree of pleomorphism they are graded as low grade to high grade tumours. *Gross:* Large capsulated area with cystic spaces and cut surface shows soft, brownish, cystic areas. *Microscopy:* It contains cystic spaces with leaf-like projections, hence the name. Cells show hypercellularity and pleomorphism. It may be a variant of intracanalicular fibroadenoma of breast.

Clinical features: They occur in premenopausal women (30-50 years). It is usually unilateral, grows rapidly to attain a large size. Swelling is *smooth, non-tender, soft, fluctuant with necrosis of skin* over the summit due to pressure. Probing can be done easily between tumour and skin across the necrosed summit (**Figs 16.53 and 16.54**). Recurrence is common.

Investigations: US; FNAC; Chest CT.

Traumatic Fat Necrosis

It may be due to either direct or indirect trauma (most often trauma may not have been noticed or forgotten).

Pathogenesis: Capillary ooze causes triglyceride in the fat to dissociate into fatty acids. It combines with



Fig. 16.53: Cystosarcoma phylloides of left breast. Note the dilated veins. Tumour occupies the entire breast.

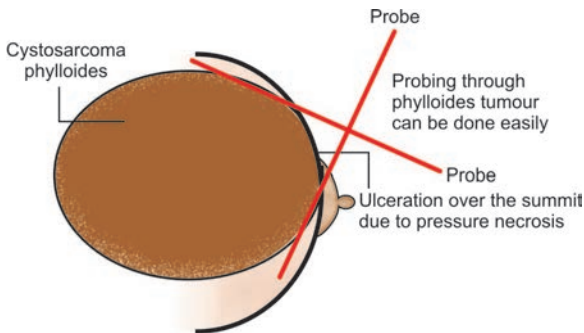


Fig. 16.54: Pressure necrosis of the skin over the summit can occur in cystosarcoma but tumour is not adherent to the skin. Probing can be done freely between tumour and under the skin.

calcium from the blood resulting in saponification which causes inflammatory reaction and later presents as a nonprogressive swelling in the breast.

Features: Painless swelling in the breast which is *smooth, hard, nontender and adherent* to breast tissue (D/D-Carcinoma). It is nonprogressive. FNAC shows chalky fluid with fat globules. Mammography is done to rule out malignancy. It often mimics carcinoma breast. Excision biopsy confirms the diagnosis and cures the disease.

Galactocele

It is seen in lactating women (can develop even 6-10 months after caseation of breastfeeding). It is

due to the blockage of lactiferous duct resulting in enormous dilatation of lactiferous sinus. It contains milk within. It is a retention cyst due to blockage of single duct which begins under the areola.

Features: Lump in the lower quadrant of the breast which is usually *unilateral, large, freely mobile, soft, fluctuant, with smooth surface and nontender*. It is a retention cyst—subareolar type. It may get *precipitated, inspissated*, or get *calcified*. When it gets calcified it mimics carcinoma breast. If it gets infected it will form an abscess (**Fig. 16.55**). US and FNAC are used to diagnose.

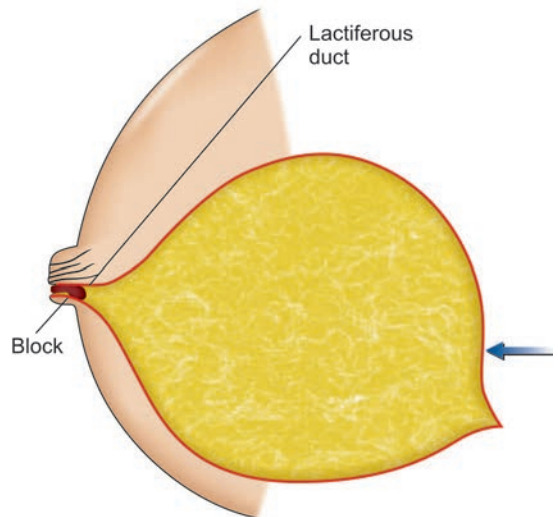


Fig. 16.55: Galactocele. Note the block at the opening of the duct.

Mastitis

It is infection and inflammation of breast tissue.

Types: Subareolar; Intramammary; Retromammary (Submammary).

Subareolar mastitis: It is the infection under the areola due to cracks in the nipple or areola. Red, inflamed, edematous areola with tender swelling underneath is the presentation. Differential diagnosis is Paget's disease of the nipple.

Intramammary mastitis: Infection occurs within the breast tissue.

a. Lactational abscess of the breast—It is commonly seen in lactating women.

Mode of infection: Bacteria (*Staphylococcus aureus*) enter the breast during sucking through the cracked nipple. Occasionally it can be of haematogenous origin. Gram negative and other bacterial infection can supervene later.

Features: Pain in the breast and fever; diffuse redness, tenderness, and induration in the breast; purulent discharge from the nipple; entire breast may get involved eventually. Differential diagnosis is inflammatory carcinoma of breast (**Figs 16.56A and B**).



Figs 16.56A and B: Typical look of breast abscess/mastitis.

Treatment: Antibiotics; Drainage under general anaesthesia, a counter incision may be needed; It is not advisable to wait till the formation of abscess.

Complications: Antibiotoma formation; sinus formation; recurrent infection.

b. Non-lactational abscess of the breast: It commonly occurs in duct ectasia and periareolar infections. Common organisms are *Bacteroides*, anaerobic streptococci, enterococci and gram negative organisms. It is commonly recurrent with tender swelling under the areola.

Retromammary mastitis: It is due to tuberculosis of the intercostal lymph nodes or ribs beneath or suppuration in the intercostal lymph nodes. It could be tuberculous disease of these tissues also. Here breast is normal. Investigations: Chest X-ray, FNAC, ESR, peripheral smear, CT chest.

Antibioma

If intramammary mastitis is not drained but only treated by antibiotics, pus localizes and becomes sterile (flaques) with a thick fibrous tissue cover, which is called as antibioma.

Features: There is previous history of mastitis treated with antibiotics. Presents with a painless swelling which is, smooth, nontender, hard, fixed to breast tissue without involving the pectorals and chest wall. Differential diagnosis is carcinoma breast (Scirrhous carcinoma breast). Investigations are FNAC, mammography and US breast.

Mastitis

Mastitis of infancy (*Witches milk*) is due to maternal hormone in infant blood. It usually subsides but may cause suppuration.

Mastitis of puberty is common in boys, is invariably unilateral with tender, swollen and inflamed breast.

Mastitis of mumps is usually unilateral and can occur in both sexes.

Bacterial mastitis is seen in adult women commonly lactating and is due to staphylococci infection.

Subareolar mastitis is due to infection of gland of Montgomery or due to areolar furuncle.

Duct Ectasia

It is dilatation of lactiferous ducts due to muscular relaxation of duct wall with periductal mastitis (*plasma cell mastitis*). Many ducts are commonly involved. It shows greenish discharge from the nipple; indurated mass under the areola which is often tender; retraction (*slit-like*) of nipple occurs at later stage of the disease;

eventually it forms an *abscess, and fistula*; often they are bilateral and multifocal; differential diagnosis is carcinoma breast. Investigations are—*discharge study* and mammography. Treatment is cone excision of involved major ducts (*Hadfield operation*) with *antibiotics*.

Mondor's Disease

Mondor's disease is thrombophlebitis of the superficial veins of the breast and anterior chest wall; presents as a thrombosed subcutaneous cord which is attached to the skin; it is often a self limiting disease without any recurrence, complication or deformity; it mimics the lymphatic permeation of carcinoma breast.

Duct Papilloma

It is usually single, from a single lactiferous duct. It is the commonest cause of nipple discharge; causes ductal dilatation by blocking the duct.

Features: Papilliferous swelling (projection), usually near the nipple orifice (4-5 cm from orifice); blood stained discharge from the nipple is common; but serous or serosanguinous discharge can also occur. Single papilloma is not premalignant. But multiple papillomas in many ducts can be premalignant. Study of discharge and ductogram may be needed (**Fig. 16.57**).



Fig. 16.57: Papilloma of breast. It is the commonest cause of bloody discharge from the nipple.

Galactorrhoea

It is secretion of milk not related to pregnancy or lactation (In true galactorrhoea milk should contain lactose, fat and milk specific proteins).

Primary galactorrhoea is due to stress and other factors. Reassurance is the treatment.

Secondary galactorrhoea is due to enhanced dopamine activity by drugs (haloperidol, methyl dopa, chlorpromazine, metoclorpramide), hyperprolactinaemia due to tumours.

Witch milk is secretion of milk in both male and female infants due to maternal hormonal effects in foetus which lasts for 3 weeks after child birth.

Gynaecomastia

It is hypertrophy of male breast more than normal, often attaining features of female breast. *Puberty* hypertrophy occurs in adolescents; *senescent* hypertrophy occurs in adult after 50 years. There is increase in ductal and/or connective tissue components of breast. It can be unilateral or bilateral (**Figs 16.58A and B**).

Presentations: Diffuse enlargement of breast occurs occupying all quadrants or as a well localised, small, firm or hard nodule under the areola which is often painful and tender.

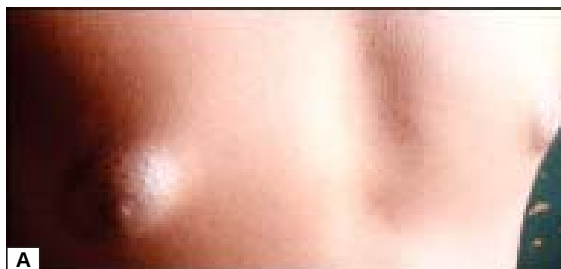
Causes: Idiopathic; Teratoma testis; Ectopic hormonal production in bronchial carcinoma; Anorchism, after castration; Adrenal and pituitary disease; Leprosy, because of bilateral testicular atrophy; Drugs: Stilboestrol, digitalis, cimetidine, spironolactone; Liver diseases and liver failure; Klinefelter's syndrome (XXY Trisomy). Rarely *gynaecomastia* may turn into *carcinoma*.

Mastalgia

It is pain in the breast. *Types:* Cyclical; Noncyclical (see Table 16.1).

Tuberculosis of the Breast

It is relatively rare; usually associated with active pulmonary tuberculosis; presents as a swelling in the breast with cold abscess, sinuses and a typical bluish appearance of surrounding skin with matted lymph nodes in the ipsilateral axilla (**Fig. 16.59**).



Figs 16.58A and B: Gynaecomastia – right side breast in a young male. Compare to opposite side to note the difference in size. Bilateral gynaecomastia in another old man is also shown.

Differential diagnosis: Carcinoma breast.

Investigations: FNAC; Frozen section biopsy is useful to differentiate from carcinoma. Excision biopsy is often needed.

Tietze's Syndrome

It is costochondritis of 2nd costal cartilage commonly seen in females, mimics mastalgia.



Fig. 16.59: Tuberculosis of breast. Mastectomy should not be done in tuberculosis. Disease often clinically mimic carcinoma breast.

Congenital Anomalies of Breast

Absence of breast is called as *amazia/amastia*. It may be unilateral or bilateral. Absence of breast; absence of sternal portion of pectoralis major muscle; usually seen in males—called as *Pendred's syndrome*. Accessory breasts (*Polymazia*) and nipple (*polythelia*, *supernumerary nipples*) can develop in axilla, groin and thigh (along the milk line). It may be unilateral or bilateral. It is due to failure of the disappearance of milk line which occurs during developmental period in normal individual. These accessory breasts may secrete milk during lactation. Accessory breast tissue is common in the axilla above the normal breast tissue. True polythelia is more than one nipple to one breast. Accessory nipple may be rudimentary and often looks like a mole. Diffuse hyperplasia; pendulous breasts; underdeveloped breasts are other anomalies which cause cosmetic challenge (**Figs 16.60 and 16.61**).

Table 16.1: Types of mastalgia

<i>Cyclical mastalgia</i>	<i>Noncyclical mastalgia</i>
• Pain related to menstrual cycle • Seen in ANDI like fibrocystadenosis • Treatment is like for fibrocystadenosis	• Pain due to causes other than ANDI like periductal mastitis, malignancy, cervical root pain, musculoskeletal pain, previous surgery, <i>Tietze's syndrome</i> (costochondritis of 2nd costal cartilage, commonly seen in females) • It is unilateral, chronic, burning / dragging pain occurs in pre- and postmenopausal age group



Fig. 16.60: Accessory nipple.



Fig. 16.61: Unilateral diffuse enlargement of breast.
Note the dilated veins on the breast.

Mammary Fistula of Atkins

It is a fistula from lactiferous duct to areolar skin. Initially recurrent abscess forms underneath the areola which points on the surface through areola causing

discharging fistula. Condition should be differentiated from carcinoma breast by discharge study, biopsy, and mammography.

Examination of Hernia

Hernia is an important clinical topic for undergraduates as well as postgraduate students in surgery. It is a long case for undergraduate students and a short case for postgraduate students in surgery. It is one of the commonest surgical entities that surgeons come across and so detail knowledge of the subject is mandatory to both undergraduates and postgraduates. Writing a case sheet for hernia is important as a long case. *Hernia means* - 'to bud' or 'to protrude' in Greek; 'to rupture' in Latin.

Hernia is protrusion of entire or part of the viscus through the wall that contains it. Herniation can occur from any cavities or through any defects, congenital or acquired. Inguinal, femoral, umbilical, incisional and epigastric are common hernias; obturator, gluteal, lumbar or Spigelian are rare hernias. Inguinal hernia is the commonest abdominal hernia. It occurs through inguinal canal. Indirect hernia comes through deep ring along the cord; direct from posterior wall of inguinal canal. Usually indirect hernia is of congenital origin, occurs in a preexisting processus vaginalis sac but often revealed later by some precipitating factors whereas direct hernia is always acquired due to weakening of posterior wall of the inguinal canal. Inguinal hernia initially is incomplete but becomes complete once it descends up to the *bottom of the scrotum*.

History taking begins with:

Name:

Age:

Sex:

Occupation:

Elderly people are more prone for hernia. Men with strain full occupation like manual labourers, sportsmen, weight lifters, etc. are more prone for hernia. Indirect hernia occurs in young; direct hernia occurs in old. Strenuous workers; gymnastics; sportsmen; weight lifters may develop hernia due to straining.

Chief Complaints

Swelling in the groin, right or left or both sided— durations; or swelling in right/left/both inguinoscrotal region for— durations.

Pain over the swelling for—durations.

History

History of Present Illness

Swelling: Duration of the swelling; mode of onset of the swelling—spontaneous or on straining should be asked. Site of first appearance of the swelling—in the groin or in the scrotum should be asked. Inguinal hernia begins in groin whereas hydrocele is purely scrotal begins in scrotum. Femoral hernia begins below the groin crease line. Progress and extent of the swelling, whether it only limits to the groin or extends down to the scrotum; or any changes in the size and extent of the swelling on standing/walking/straining/lying down should be asked. Whether swelling is reducible on lying down/partially reducible or irreducible on lying down or needs any manoeuvre to reduce it should be also asked. History of gurgling sound in the scrotum signifies enterocele. In irreducible swelling one should be asked for the history of pain, any abdominal distension/vomiting.

Pain: Site of pain—whether it is in the groin or in the scrotum; duration of pain; severity of the pain; type of pain—dull aching or severe pricking type; aggravating or relieving factors should be asked. Pain may be aggravated on straining/walking/weight lifting; relieved on lying down. Patient with hernia often may feel dragging or aching pain in the groin more after straining prior to development of swelling in the groin. Pain in an existing hernia may be due to drag on

omentum, mesentery, adhesions, inflammation, obstruction or strangulation. Obstruction causes distension and vomiting. Strangulation causes severe tenderness and toxicity.

History relevant to precipitating factors: Chronic cough, tuberculosis, bronchial asthma or other respiratory diseases; constipation, altered bowel habits, tenesmus, bloody stool—in relation to anorectal stricture/carcinoma; dysuria/urgency/hesitancy/altered stream/night frequency/retention of urine/burning urine/haematuria—in relation to benign prostatic hyperplasia/urethral stricture.

History suggestive of complications: Irreducibility, severe pain in the groin over the swelling and also colicky abdominal pain, abdominal distension, vomiting, and constipation should be asked.

Past History

Past history of hernia surgery—same side/opposite side. Type of surgery whether mesh used or repair done. History of appendicectomy done earlier (ilioinguinal nerve may be injured causing direct hernia) and if so details about the surgery (can cause right sided direct hernia). Past history suggestive of irreducibility/obstruction and treatment received for that whether conservative/surgical should be asked.

Personal History

History of smoking—duration, number per day, whether beedi or cigarette should be asked. History of pan chewing/alcohol intake; appetite and altered weight should be asked.

General Examination

Examination is done for general built and nutritional—status, pallor, clubbing, cyanosis, jaundice, lymphadenopathy, and oedema feet. Pulse and blood pressure is recorded.

Local Examination

Inguinoscrotal region should be examined in standing position as swelling commonly reduces and disappears in lying down position. Area from umbilicus to mid-thigh region should be exposed after taking consent for examination (**Fig. 17.1**).



Fig. 17.1: All hernias should be inspected initially on standing.

Inspection

Inspection is done always *first in standing* straight up without bending *later in lying down* position.

Inspection in standing position

Side of the swelling should be observed and mentioned. Extent of the swelling is important. Incomplete indirect inguinal hernia and usually direct inguinal hernias are located in inguinal region. Complete indirect inguinal hernia (rarely complete direct inguinal hernia) is inguinoscrotal—extending down into the bottom of the scrotum (**Fig. 17.4**). Swelling extends from the proximal part of the inguinal canal towards the scrotum below. Both transverse and vertical dimensions of the size should be mentioned. Shape of the swelling is *pyriform* in indirect inguinal hernia and *globular/hemispherical* in direct inguinal hernia or femoral hernia.

Expansile impulse on coughing over the swelling is diagnostic. It is better seen than felt. While patient is in standing position, and examiner sits beside the



Figs 17.2A and B: Expansile impulse on coughing is better seen than felt. It should be inspected with patient standing and examiner sitting beside the patient.

patient and is asked to turn his face to opposite side (to prevent coughing towards examiner) and cough. Expansile impulse is visible in the groin area; or an already existing swelling will become much more prominent as the intestines or omentum (contents) gets driven into the hernial sac. Often swelling appears only during the act of coughing and disappears later. Absence of expansile impulse does not rule out the possibility of hernia (**Figs 17.2A and B**).

Surface—Smooth/uneven; **Margin**—well-defined/ill-defined; **Visible peristalsis** over the swelling should be noted if present (should not be mistaken for dartos muscle contraction). It means it could be enterocoele. **Scar/dilated veins/discolouration/redness** over the swelling should be noted. On inspection, whether testis is seen separately from the swelling or covered by the swelling all over should be noted.

Skin over the swelling is usually normal. Scar of recurrent hernia may be evident. Type of scar, linear or wide; healed by primary or secondary intention should be assessed. Infected wound causes wide deep

puckered scar which may be the cause for recurrence. Skin may be stretched. Atrophy of skin can occur. In strangulated hernia skin may be oedematous and red. Wearing hernia truss may cause haemosiderin laid brown pigmentation of skin. Occasionally strangulated hernia may cause ulceration due to skin necrosis.

The features of hernia

- Expansile impulse on coughing
- Reducibility of the content on lying down or by direct pressure.

Note: These two features may be absent once hernia is strangulated.

Palpation

Temperature and tenderness over the swelling (in strangulated/inflamed hernia) is noted.

Whether it is possible to *get above the swelling* or not—one can get above purely scrotal swelling but not in inguinoscrotal swelling. It is checked in *standing* position.

Position and extent of the swelling; Size in vertical and transverse directions; Margin well defined or ill defined; Surface smooth/lobular/tense; Consistency—soft and elastic in enterocoele; doughy in omentocoele (epiplocele) should be noted.

Location of the swelling—swelling is above and medial to pubic tubercle in inguinal hernia and below and lateral to pubic tubercle in femoral hernia. Pubic tubercle may be reached by following the tendon of adductor longus. In obese patients often it may be difficult to differentiate between inguinal and femoral hernia. Occasionally femoral hernia may ascend upwards from saphenous opening superficial to inguinal ligament to present as swelling in inguinal region which is invariably irreducible.

Reducibility of the swelling is checked by different methods. Whether it reduces spontaneously while lying down (usually direct hernia) and gets reduced completely or partially should be checked. Patient himself reduces the content easily if asked. In enterocoele it is difficult to reduce the first part but last part gets reduced easily. In omentocoele it is difficult to reduce the last part but first part gets reduced easily. Whether swelling needs any manipulation to get reduced like *taxis* is to be noted. *Taxis* is *gradual reduction of contents of the scrotum by gentle*



Fig. 17.3: Inguinal hernia is reduced in lying down position; done by elevating the scrotum and flexing and rotating the hip—*taxis*.

manipulation by flexion, adduction and rotation of hip joint. This maneuver relaxes the superficial ring and oblique abdominal muscles (**Fig. 17.3**). Fundus of the sac is held with one hand and contents are gently squeezed towards the abdomen and other hand guides the content across superficial inguinal ring.

Expansile impulse on coughing also should be checked during palpation in *standing* position. After complete reduction of the contents of the swelling, either by placing finger on superficial ring or by holding the root of the scrotum between index and thumb, patient is asked to cough to feel expansile impulse on coughing. Fingers may get separated allowing contents to force down. Impulse will be absent in strangulated hernia, incarcerated hernia, in presence of adhesions blocking the entrance of sac.

Zieman's test is done to find out over which finger cough impulse is felt and so which type of hernia it could be—whether femoral/direct inguinal or indirect inguinal.

Deep ring occlusion test: When deep ring is occluded after reducing the contents, if impulse on coughing is absent in standing position then it is indirect inguinal hernia; if impulse on coughing is still present then it is direct inguinal hernia.

Finger invagination test: Size of the superficial ring is noted and site of the impulse felt is observed whether it is in the tip of the finger or on the pulp.



Fig. 17.4: Giant hernia. Note that bottom of the hernia in the scrotum is below the level of middle of the thigh (scrotal abdomen). Note also the burial of penis. Such patient is difficult to manage as often the contents cannot be reduced completely into the abdominal cavity during surgery as peritoneal cavity is reduced in size; if done forcibly it results in abdominal compartment syndrome with IVC compression and diaphragmatic elevation.

Palpation of testis, epididymis and spermatic cord should be done without fail. Relation of swelling to testis also should be noted.

Opposite inguinal region, opposite testis, epididymis and spermatic cord should be examined. Presence or absence of impulse on coughing on opposite side should be mentioned.

Bulbar urethra is palpated by lifting the scrotum and feeling in the midline (To look for thickening and button-like depression—a feature of stricture urethra) (**Fig. 17.5**).



Fig. 17.5: Bulbar urethra should be palpated by raising the scrotum in midline posteriorly. Any stricture urethra is felt as thickening/button-like depression. Gonococcal urethritis and trauma are the commonest causes of stricture urethra. Bulbar urethra is the commonest site of stricture urethra.

Percussion

Without reducing contents of the swelling, percussion is done over the surface. If it is resonant, it is enterocele. If it is dull on percussion then it is omentocele.

Auscultation

Bowel sounds may be heard over the swelling if it is enterocele.

Per Abdomen Examination

Abdominal muscle tone should be checked by shoulder and head raising test, leg raising test and Valsalva maneuver. It should be inspected for *Malgaigne bulging* and should be palpated to check whether the tone is adequate (firm) or inadequate (supple).

Any scar over the abdomen (appendicectomy scar may cause right sided direct inguinal hernia); ascites or mass per abdomen should be mentioned (**Figs 17.6A to C**).



Figs 17.6A to C: Head and shoulder raising and Valsalva manoeuvre tests are needed to check the tone of abdominal muscle in hernia.

Digital Examination of the Rectum (P/R)

Rectal examination is done in all hernia cases to look for prostate enlargement in elderly and rectal/anorectal strictures. Causes of rectal stricture are – recurrent proctitis, ulcerative colitis, carcinoma, previous anal surgery, LGV induced proctitis, tuberculosis, etc. (Figs 17.7 and 17.8).



Fig. 17.7: Clinically per-rectal examination is a must in hernia to look for prostate enlargement, and rectal stricture which are precipitating factors.



Fig. 17.8: Inguinal hernia in a patient who is having benign prostatic hyperplasia (BPH) with Foley's urinary catheter inserted. He is also having paraphimosis and so not able to replace back the retracted prepuce after catheterisation.

Examination of Respiratory System

Respiratory system is examined for altered breath sounds (rhonchi, bronchial breathing), effusion, etc. to find out any precipitating causes like tuberculosis, bronchitis, asthma, bronchiectasis (Fig. 17.9).

Other Systems

Cardiovascular system, nervous system including spine and cranium are examined for any neurological problems before management of hernia.



Fig. 17.9: Respiratory system should be examined to find out the precipitating causes for hernia like bronchitis, tuberculosis or asthma.

Investigations

All case sheets for long case should mention the investigations required for that particular case.

Relevant investigations required for inguinal hernia are chest X-ray, haematocrit, blood sugar, serum creatinine, ultrasound abdomen depending on the age/suspected cause for the hernia. Chest X-ray is done to look for bronchitis, tuberculosis, bronchiectasis. US abdomen is done to look for benign prostatic hyperplasia, residual urine, ascites, and mass lesion.

Note: Presentation of the case should be in order as mentioned above. One cannot alter the order of presentation like presenting percussion first and later palpation or likewise in a haphazard manner. Students should strictly follow the proper order of presentation in clinical methods.

Why clinically it is called inguinal hernia?

Patient presents with a swelling in the groin, which has gradually increased in size, often descends into the scrotum and gets reduced on lying down. It increases on straining, coughing or walking. Expansile impulse on coughing is present and reduces on lying down or by taxis.

Why it is indirect inguinal hernia?

It is pyriform in shape. It descends obliquely in the groin. On occluding the internal ring in *ring occlusion test*, swelling does not appear later on coughing. On *ring invagination test*, impulse is felt at the tip of the invaginating finger. *Zieman's test* confirms the impulse over the index finger.

If it is direct inguinal hernia what are the differentiating features?

Direct inguinal hernia is globular in shape. After occluding the deep ring, swelling still appears on the medial side of the inguinal region on coughing. Impulse is felt on the pulp of the finger in invagination test and over the middle finger in Zieman's test.

How expansile impulse on coughing is clinically demonstrated?

Expansile impulse on coughing is seen on inspection when patient is asked to cough. Expansile impulse on coughing is also felt by placing the thumb in front, middle and index fingers behind the root of the scrotum and asking the patient to cough.

When in a hernia impulse on coughing will not be present?

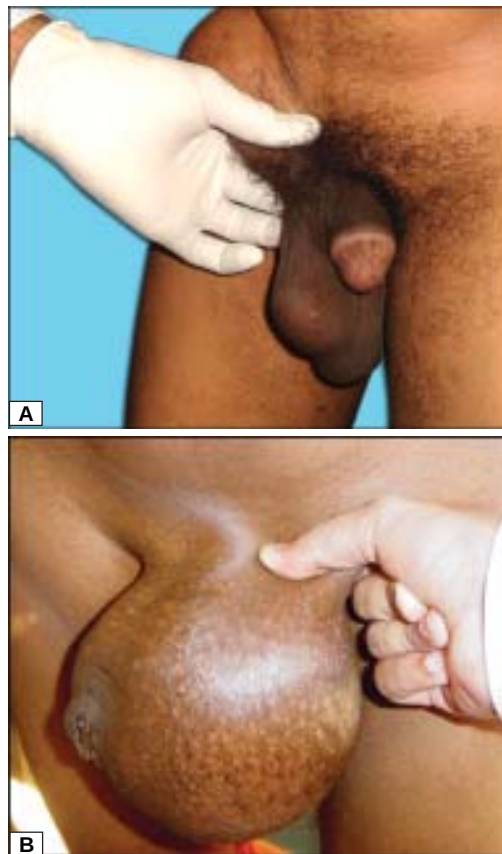
Strangulated hernia will not show impulse on coughing.

What is the meaning of 'get above the swelling'?

Root of the scrotum is held between the thumb in front, index and middle fingers behind. In purely scrotal swelling like vaginal hydrocele, fingers and thumb can be approximated well without any additional structures other than cord in between (*one can get above the swelling*). In case of inguinoscrotal swelling thumb and fingers do not meet each other properly because of the descent of hernial contents down (*one cannot get above the swelling*). It occurs in funicular and complete type of inguinal hernia but not in bubonocoele (**Figs 17.10A and B**).

What is ring occlusion test?

It is the most important test in inguinal hernia. It is performed in *standing* position. Deep/internal ring is located 1.25 cm above the mid-inguinal point. Mid-inguinal point is mid-point between the anterior superior iliac spine and pubic symphysis (*Note: Mid-point of the inguinal ligament is centre point between anterior superior iliac spine and pubic tubercle*). Patient



Figs 17.10A and B: In inguinoscrotal swelling one cannot get above the swelling. In scrotal swelling one can get above the swelling. It is convenient and easier to use left hand for right side inguinoscrotal / scrotal swelling and right hand for left side to check the 'get above the swelling' even though any hand can be used.

is asked to lie down to reduce the hernial contents. Thumb is placed over the mid-inguinal point. Patient is asked to cough. If there is expansile impulse on coughing on the medial side of the thumb, even after deep ring occlusion, it is then direct inguinal hernia. If there is no impulse on coughing then patient is asked to stand with thumb occluding the deep ring. Patient is once again asked to cough; impulse on the medial side of the occluded thumb is looked for to rule out the direct inguinal hernia. If there is no impulse even on standing, it is indirect inguinal hernia. The occluded thumb is removed and patient is asked to cough to show the swelling and impulse due to indirect inguinal hernia (**Figs 17.11A and B**).



What is the prerequisite for ring occlusion test?

Hernia should be reduced completely prior to do deep ring occlusion test. One cannot do deep ring occlusion test/invagination test/Zieman's test if hernia is irreducible.

How is finger invagination test done?

Patient is asked to lie down. Contents are reduced completely. Using the little finger, scrotal skin is invaginated from below upwards near upper part of the testis. Finger is reached towards the superficial inguinal ring/external ring. Normally external ring does not admit the tip of the little finger. Finger is rotated inwards so that nail is towards the cord side and pulp is towards the ring. Right hand is used for right side and left hand for left side. Patient is asked to cough. If the impulse is felt on the tip of the finger, then it is indirect inguinal hernia. If impulse is felt on the pulp then it is direct inguinal hernia. In case of complete inguinal hernia or funicular hernia external ring is patulous which can be very well assessed by

Figs 17.11A and B: Ring occlusion test is done to find out whether hernia is direct or indirect. If after occluding the ring swelling appears on the medial side, it is direct hernia. If swelling does not appear on occlusion and coughing it is invagination test. Index finger can also be used for the test. In direct hernia finger goes directly; in indirect hernia finger goes upwards and outwards. One should also remember that patulous wider ring does not mean that patient should always have hernia (**Figs 17.12A and B**).

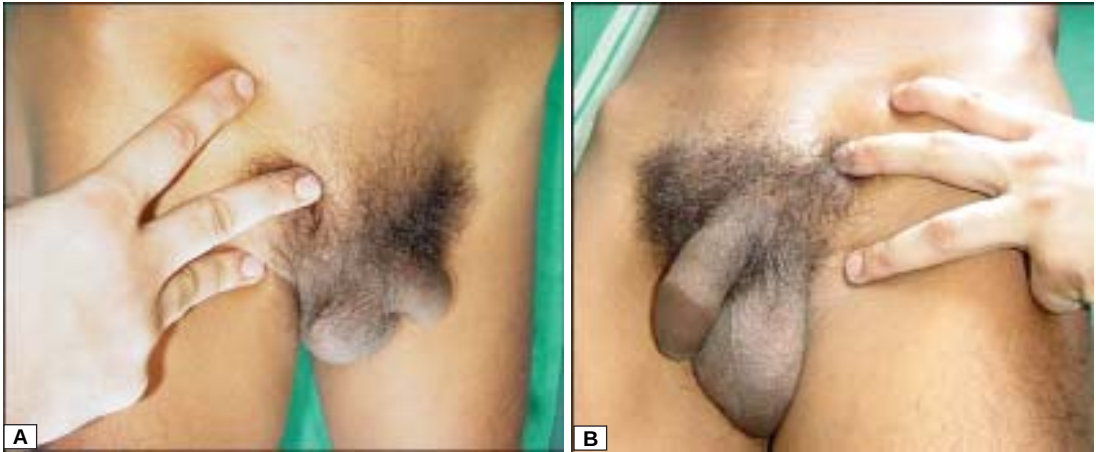
Invagination test: Index finger is invaginated across scrotum towards the external ring. When patient coughs, inguinal hernia is felt as a slit-like sensation.

Invagination test should be done very gently, otherwise it will be very painful. It cannot be done in children.

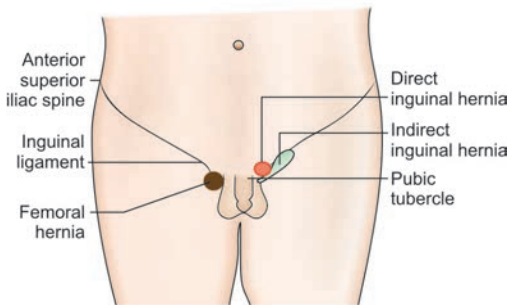
Silk glove sign: Index finger is invaginated across scrotum towards the external ring. When patient coughs, inguinal hernia is felt as a slit-like sensation.

How is Zieman's test done?

The hernial contents are reduced. Index finger is placed over the deep ring. Middle finger is placed over the superficial ring and ring finger over the saphenous opening. Patient is asked to cough.



Figs 17.12A and B: Little finger is used to do invagination test.



Figs 17.13A and B: Zieman's test—done on both sides. Three fingers are used to do Zieman's test.



Fig. 17.14: Anatomical location of indirect, direct and femoral hernias.

If impulse touches (**Figs 17.13A and B and 17.14**):

- *Index finger it is*—indirect inguinal hernia.
- *Middle finger it is*—direct inguinal hernia.
- *Ring finger it is*—femoral hernia.

Testing Inguinal Hernia in Children

Fullness is seen over the groin when compared to opposite side is seen. In difficult small hernia, child is made to cry or jolt or jump, later superficial ring is palpated to feel the cord which will be thicker than opposite side. Rolling the contents of the inguinal canal by finger will give the sensation of finger of a rubber glove which is wet inside (**Fig. 17.15**).

Gornall's test—Child is held from back to place both hands in front over the abdomen which is pressed with

fingers and child is lifted up. This raises the intra-abdominal pressure to make hernia more prominent (**Fig. 17.16**).

Inguinal Hernia in Females

Hernia in females is rare. Inguinal hernia is commonest type of hernia in females. Femoral hernia is common in females. *Expansile impulse on coughing* is diagnostic. Invagination test is *not possible* in females. Palpation of labium majus demonstrates *thickness* compared to opposite side indicating hernia in canal



Fig. 17.15: Right sided hernia in a child. Only herniotomy is done for inguinal hernia in children. Repair/mesh are not used. Herniotomy is also done for hydrocele in children of Nuch. Patients should be properly examined in standing position, otherwise hernia is more likely to be missed. Reducible inguinal hernia is obvious by its clinical features. But *femoral hernia* needs to be differentiated by its definitive anatomical location. *Differential diagnosis* for irreducible inguinal hernia in females—A *hydrocele of canal of Nuck* is smooth, fixed, fluctuant and brilliantly transilluminant swelling. *Bartholin cyst* is confined to labium majus; one can get above the swelling; it does not extend to superficial inguinal ring; it is not transilluminant (**Fig. 17.17**). *Groin abscess* is smooth, soft, fluctuant and tender; but it is often difficult to differentiate it from strangulated hernia. Associated abdominal symptoms favors strangulated inguinal hernia.

How inguinal hernia is differentiated from femoral hernia?

Inguinal hernia is above and medial to the pubic tubercle. Femoral hernia is below and lateral to the pubic tubercle.



Fig. 17.16: Gornall's test.

What is taxis?

Taxis (taxis means arrangement) is a method used to reduce a complete inguinal hernia. Hip and knee are flexed and thigh is adducted. One hand is held near the fundus of the sac in the bottom of the scrotum, other hand placed adjacent to external ring, and contents are gently reduced towards the proximal side. Often patient can himself do this technique in a better way. It is contraindicated in obstructed/strangulated hernia or femoral hernia or Maydl's hernia. Taxis should be done very gently.

How is tone of abdominal muscle checked and why?

Abdominal muscle tone is checked by head and shoulder rising (without supporting the elbows) or leg rising tests. It is initially inspected for any bulges in the abdominal wall which signifies *Malgaigne bulgings*. Later abdomen should also be palpated for muscle tone. Firmness signifies adequate tone whereas suppleness signifies poor muscle tone. Poor muscle tone indicates that patient needs hernioplasty using mesh. Abdominal muscle tone is also checked by Valsalva maneuver.

'Use five fingers of the hand to complete all tests for hernia'.

- Thumb—for deep ring occlusion test.
- Index, middle and ring fingers for Zieman's test.
- Little finger for superficial ring invagination test.

Rules of hernia examination

- Never forget to check expansile impulse on coughing and reducibility.
- Never forget to examine opposite side.
- Never forget to do per-rectal examination.
- Never forget to examine bulbar urethra.
- Never forget to check abdominal muscle tone.

Expansile impulse on coughing

- Hernia
- Meningocele
- Laryngocele
- Empyema necessitans
- Intracranially extended dermoid

Boundaries and Anatomy of the Inguinal Canal

In front: External oblique aponeurosis and conjoined muscle laterally.

Behind: Inferior epigastric artery, fascia transversalis and conjoined tendon medially.

Above: Conjoined muscle (Arched fibres of internal oblique).

Below: Inguinal ligament.

Superficial inguinal ring is a triangular opening in the external oblique aponeurosis and is 1.25 cm above the pubic tubercle. The ring is bounded by a superomedial and inferolateral crus. Normally the ring may just admit or may not admit the tip of little finger (**Fig. 17.18**). *Deep inguinal ring* is a U-shaped condensation of the transversalis fascia, lies 1.25 cm above the inguinal ligament midway between the

symphysis pubis and the anterior superior iliac spine. *Inguinal canal* – In infants both superficial and deep rings are superimposed without any obliquity of the inguinal canal. In adults it is 3.75 cm long, directed downwards and medially from the deep to superficial ring. In males inguinal canal transmits the spermatic cord, ilioinguinal nerve and genital branch of the genitofemoral nerve. In females, its content is the round ligament. Inguinal canal in female is called as *canal of Nuck*.

Inguinal defence mechanism: It is the natural mechanism to maintain the strength of the inguinal canal. It is by - Obliquity of the inguinal canal; arched conjoined tendon; shutter mechanism of internal oblique; ball valve mechanism of the cremaster; slit valve mechanism of the intercrural fibres of the superficial inguinal ring.

Types of Indirect Inguinal Hernia

It can be *incomplete* wherein sac does not reach to the bottom of the scrotum. It can be *complete* wherein sac descends completely up to the bottom of the scrotum. *Incomplete type* can be *bubonocoele* where hernia limits to inguinal region without passing through the superficial inguinal ring or can be *funicular* where sac reaches up to the level of the upper part of the testis into the scrotum across the external ring.

80% of inguinal hernia are indirect. Neck of the sac is lateral to inferior epigastric artery. All hernia (almost) in children and females are indirect type. After occluding internal ring content will not descend. It is commonly congenital occurring in a preformed (pre-existing) processus vaginalis. Normally funicular part of the processus vaginalis gets obliterated. In these patients it remains patent forcing herniation suddenly due to some precipitating causes. Commonly sac here is complete. Acquired indirect sac also can occur (**Figs 17.19 to 17.23**).

Direct Hernia

It is 15% of all hernias; 50% are bilateral; 35% of inguinal hernias; uncommon in females; always acquired due to weak posterior wall of inguinal canal. It occurs through Hesselbach's triangle. It is medial to inferior epigastric artery; neck is wider; sac is often thick; medial wall or content may be urinary bladder.

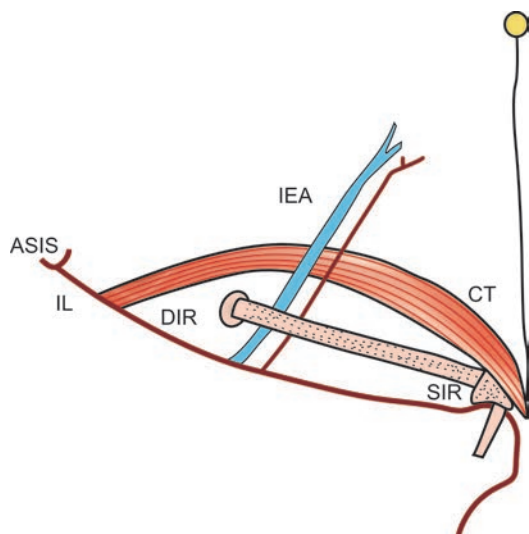


Fig. 17.17: Inguinal hernia left sided in a female. Note thick labium left side.

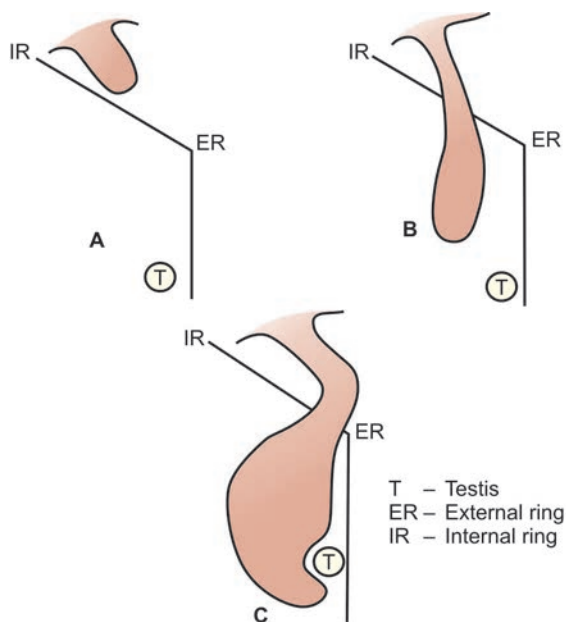


Fig. 17.18: Anatomy of the inguinal canal. IL—Inguinal Ligament. SIR—Superficial Inguinal Ring. DIR—Deep Inguinal Ring. CT—Conjoined Tendon. ASIS—Anterior Superior



Fig. 17.19: Types of indirect inguinal hernia.

(A) Bubonocoele, (B) Funicular, (C) Complete.

It is usually reducible; becoming complete by descending into the bottom of the scrotum is rare but can occur.

Hesselbach's triangle: It is bounded by inferior epigastric artery laterally, lateral border of rectus muscle medially and inguinal ligament below. Direct hernia protrudes out through this triangle. It is divided into medial and lateral by obliterated umbilical artery (**Figs 17.24A to 17.26**).



Fig. 17.20: Bubonocoele.



Fig. 17.21: Incomplete inguinal hernia – funicular type. It is probably irreducible in this patient.

Differences between enterocele and omentocoele (Figs 17.29 and 17.30)

Enterocele	Omentocoele (epiplocele)
First part is difficult to reduce but last part is easier. There will be gurgling sound on reduction	First part is easier to reduce but last part is difficult. Has a doughy feeling
Resonant on percussion	Dull on percussion
Peristalsis is seen	No peristalsis seen
Bowel sounds may be heard	Bowel sounds not heard

Differences between indirect inguinal and direct inguinal hernias (Figs 17.27 and 17.28)

<i>Indirect inguinal hernia</i>	<i>Direct inguinal hernia</i>
Can occur from childhood to adult	Common in elderly
Occurs in a pre-existing sac	Always acquired
Protrusion through the deep ring; herniation occurs later	Herniation through posterior wall of the inguinal canal
Pyriform/oval in shape; descends obliquely and downwards	Globular/round in shape; descends directly forwards as a bulge
Can become complete by descending down into the scrotum	Descent down into the scrotum is rare
Neck of the sac is narrow and lateral to inferior epigastric artery	Neck of the sac is wide and medial to inferior epigastric artery
Sac is anterolateral to the cord	Sac is posterior to the cord
Ring occlusion test does not show any impulse after occluding the deep ring	Test shows impulse even after occluding the deep ring
Invagination test shows impulse on the tip of the little finger	Impulse is felt over the pulp of the little finger
Zieman's test shows impulse on the index finger	Test shows impulse on the middle finger
Commonly unilateral but can be bilateral	Commonly bilateral
Obstruction/strangulation are common	Rare but can occur
Sac should be opened during surgery	Sac is not necessarily opened unless obstruction is present

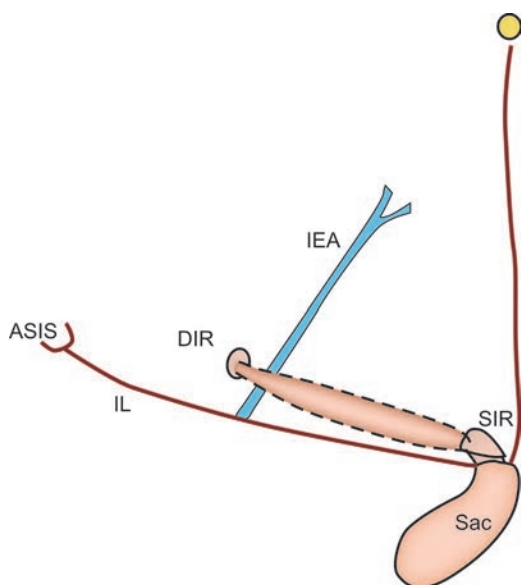


Fig. 17.22: Complete inguinal hernia is one where hernia descends completely into the scrotum.

Differential Diagnoses for Groin Swelling (Figs 17.31 and 17.32)

Indirect/direct inguinal hernia: Swelling is above and medial to pubic tubercle; expansile impulse on coughing; reducibility are the features.

Hydrocele—Vaginal/encysted: One can get above the swelling; absence of expansile impulse on coughing; fluctuation is positive. Hydrocele of the canal of the Nuck in females is transilluminant.

Femoral hernia: Swelling is below and lateral to pubic tubercle with impulse on coughing.

Lipoma of the cord: Swelling in the inguinal canal without any impulse on coughing. It is often observed that hernia and lipoma of the cord can coexist and is identified only on table during surgery.

Inguinal lymphadenopathy: Palpable inguinal nodes may be of vertical or horizontal group in the inguinal canal may be due to non-specific causes or filarial lymphadenitis or secondaries in inguinal nodes primary being in the limb or perineum or lymphoma.

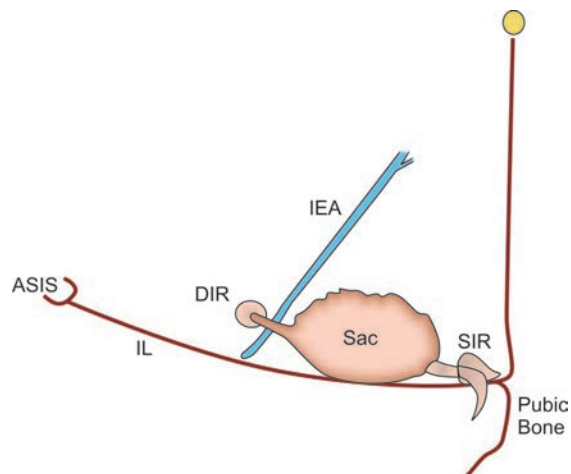


Fig. 17.23: Diagram of indirect inguinal hernial sac. IL—Inguinal Ligament. SIR—Superficial Inguinal Ring. DIR—Deep Inguinal Ring. ASIS—Anterior Superior Iliac Spine. IEA—Inferior Epigastric Artery.

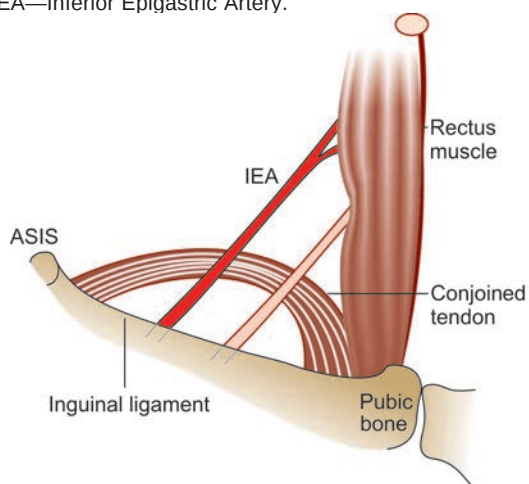


Fig. 17.25: Anatomy of Hesselbach's triangle. ASIS—Anterior Superior Iliac Spine. IEA—Inferior Epigastric Artery.

Figs 17.24A and B: Direct hernia arises through Hesselbach's triangle. IL—Inguinal Ligament. SIR—Superficial Inguinal Ring. DIR—Deep Inguinal Ring. ASIS—Anterior Superior Iliac Spine. IEA—Inferior Epigastric Artery.

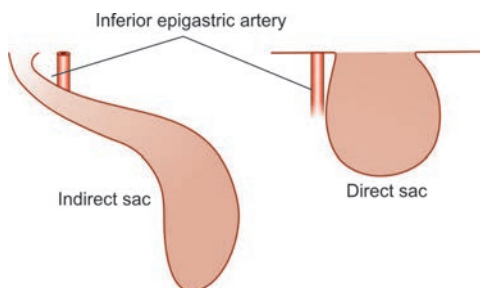


Fig. 17.26: Large bilateral direct hernias. Note, on right side it has descended into the scrotum to become complete.

Usually direct hernia will not descend into the scrotum but long standing direct hernia can descend down and become complete.

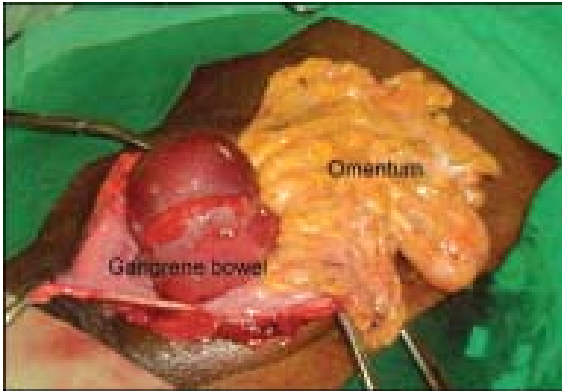


Fig. 17.27: Diagrammatic representations of direct and indirect sacs.

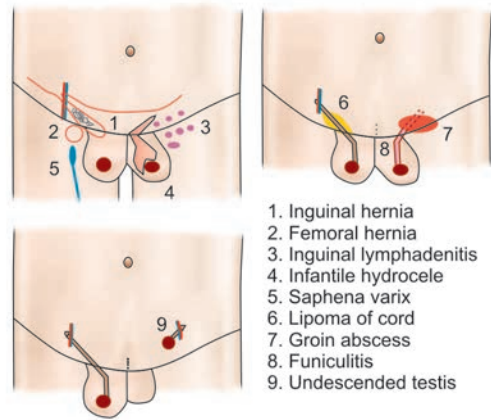


Fig. 17.29: Irreducible hernia with bowel as well as omentum as contents. Note the change in color of the bowel.

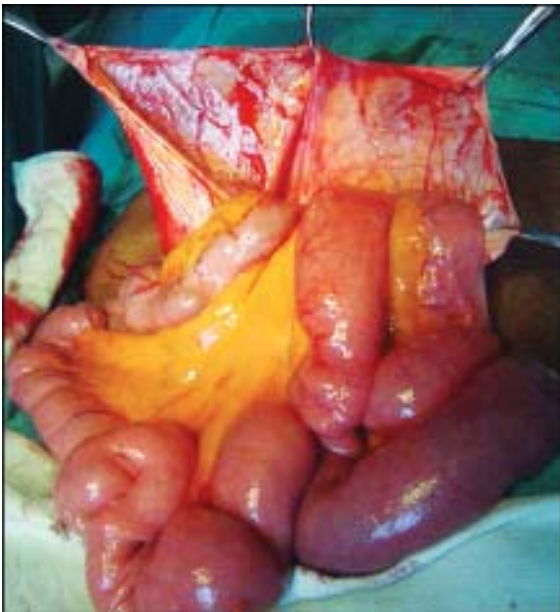


Fig. 17.28: Bilateral direct hernia. Note the medial location of the hernia. Direct hernia occurs through Hesselbach's triangle.



Fig. 17.30: Hernial sac with small bowel (enterocele) as content.

Groin abscess: Fluctuant smooth, soft, tender, non-mobile swelling in the groin could be an abscess due to lymph node suppuration. It is often difficult to differentiate it from strangulated hernia.

Undescended testis: It presents as firm swelling in the inguinal region; associated commonly with indirect hernia. Testicular sensation may be elicited. Empty scrotum is evident. It can be bilateral. *Ectopic testis* is also often located in groin.

Infantile hydrocele: It presents as swelling in the inguinal region as well as in the scrotum. Impulse on coughing will be absent as it is not communicating into the peritoneal cavity.

Parts of Hernia

It consists of neck, body and fundus. Neck is narrow in indirect sac which is obliquely placed in the inguinal

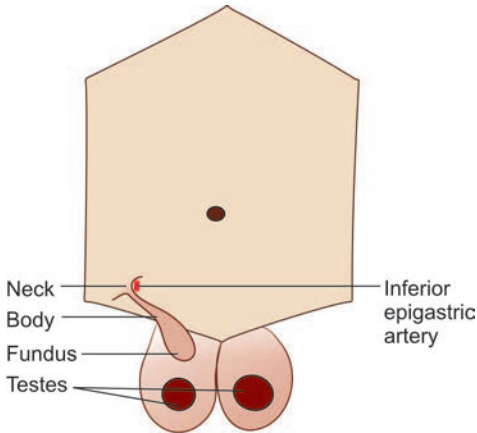


Fig. 17.31: Differential diagnosis for groin swellings.

canal (oblique hernia). Neck is wide in direct sac which is placed posteriorly, medially and directly. Neck is lateral to inferior epigastric artery in indirect sac; medial to inferior epigastric artery in direct sac. Sac is opened in the fundus in indirect sac. Sac is usually not required to be opened in direct sac unless there are adhesions (Fig. 17.33).

Groin Hernia

Groin hernia occurring through a myopectineal orifice. It can be indirect inguinal hernia/direct inguinal hernia or femoral hernia. A *hernia* is defined as an area of weakness or disruption of the fibromuscular tissues of the body wall. Hernia is also often defined as an actual anatomical weakness or defect. 75% of abdominal wall hernias are groin hernias. 15% of males and 5% of females will develop groin hernia. Presently all hernias in groin are grouped as *groin hernias*.

Fruchaud's Myopectineal Orifice

It is an osseo-myo-aponeurotic tunnel. It is bounded- medially by lateral border of rectus sheath; above by the arched fibres of internal oblique and transverse abdominis muscle; laterally by the iliopsoas muscle; below by the pectin pubis and fascia covering it. It is through this tunnel all groin hernias occur.

Newer Anatomical Considerations

Preperitoneal space is a potential space in front of the peritoneum and behind the transversalis fascia and

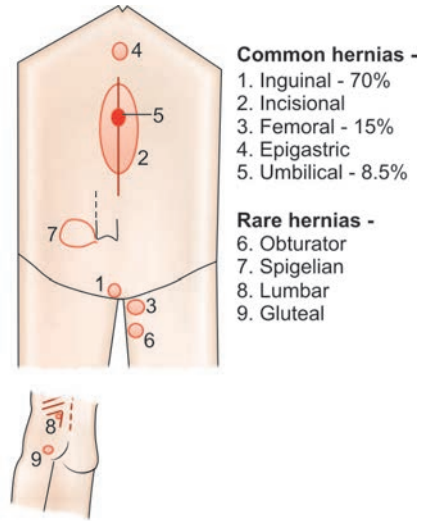


Fig. 17.32: Bilateral inguinal hernia and right sided femoral hernia. Femoral hernia is rare in males but can occur. anterior rectus muscle. Below in front of the urinary bladder it is called as *space of Retzius* (medially), laterally it is called as *space of Bogros*. Median umbilical fold is formed by urachus in the midline. Medial umbilical ligament is formed by obliterated umbilical arteries. Lateral umbilical fold by inferior epigastric vessels. Three fossae are lying in relation to these folds- *supravesical* and *medial fossae* are medial to lateral umbilical fold which are sites of direct hernia whereas *lateral fossa* is lateral to lateral umbilical fold is site of indirect hernia (Fig. 17.34).

In 1956 Fruchaud described his *myopectineal orifice* bounded medially by the lateral border of rectus abdominis, laterally by iliopsoas, superiorly by conjoint tendon and inferiorly by pectin pubis. This area is the site of groin hernia which should be covered by mesh of adequate size to strengthen the defect and to prevent the recurrence. *Iliopubic tract* is analogue of the inguinal ligament extends from Cooper's ligament to anterior superior iliac spine which divides endoscopic view of pre-peritoneal space into *superior compartment* (contains inferior epigastric artery, Hesselbach's triangle, cord structures and site of indirect inguinal hernia) and *inferior compartment* (contains femoral canal, iliac vessels, iliopsoas muscle, genitofemoral nerve, lateral femoral cutaneous nerve). External iliac vessels lie in a triangle formed by gonadal vessels laterally, vas deferens medially and peritoneal reflection inferiorly (*triangle of doom*).

Aberrant obturator artery which is an occasional branch of inferior epigastric artery replacing its pubic branch travels across Cooper's ligament, which during fixation of mesh can cause torrential haemorrhage—*circle of death*. *Triangle of pain* is formed by gonadal vessels medially, iliopubic tract laterally and peritoneal reflection below. Genitofemoral nerve and lateral cutaneous nerve of thigh traverse this triangle. Injury to these nerves either by dissection or by tack (*during laparoscopic hernia repair*) cause postoperative pain. Tacks/staplers should not be placed in this triangle.

Newer Classifications of Groin Hernias

Gilbert Classification (1987)

Type I: Hernia has got snug internal ring through which a peritoneal sac passes out as indirect sac.

Type II: Hernia has a moderately enlarged internal ring which admits one finger but lesser than two finger breadth. Once reduced it protrudes during coughing or straining.

Type III: Hernia has got large internal ring with defect more than two finger breadth. Hernia descends into the scrotum or with sliding hernia. Once reduced it immediately protrudes out without any straining.

Type IV: It is direct hernia with large full blow out of the posterior wall of the inguinal canal. The internal ring is intact.

Type V: It is a direct hernia protruding out through punched out hole/defect in the transversalis fascia. The internal ring is intact.

Type VI: Pantaloon/double hernia.

Type VII: Femoral hernia.

Type VI and VII are Robbin's modifications.

Nyhus Classification

Type I: Indirect hernia with normal deep ring.

Type II: Indirect hernia with dilated [patulous] deep ring.

Type III: Posterior wall defect.

- a. Direct hernia, sliding hernia.
- b. Pantaloon hernia.
- c. Femoral hernia.

Type IV: Recurrent hernia.

Bendavid Classification

Type I: Anterolateral defect (indirect).

Type II: Anteromedial (direct).

Type III: Posteromedial (Femoral).

Type IV: Posterior-prevascular hernia.

Type V: Anteroposterior defect (Inguinofemoral hernia).

Clinical Classification of Hernia

Reducible hernia: Here contents can be reduced to abdominal cavity but sac is in position.

Irreducible hernia: Here contents cannot be reduced to abdominal cavity. It is due to adhesion between contents/adhesion between content and sac/adhesions between surfaces of the sac; sliding hernia; large hernia complete type; narrow neck of the sac acting as a constricting band preventing reduction of the content.

Obstructed hernia: Here irreducibility causes occlusion of the lumen of the intestine but bowel remains viable.

Strangulated hernia: Here irreducibility, obstruction and compromised blood supply of the bowel occurs. Initially venous congestion occurs later arterial blood supply is also compromised causing gangrene of the bowel. Tender, tense, swelling with toxicity are the features. Strangulation without obstruction occurs in Richter's hernia, omental strangulation.

Inflamed hernia: Here hernial contents being appendix/Meckel's diverticulum/fallopian tube are inflamed or sac itself is inflamed. It is not tense. It is very rare.

Incarcerated hernia: Lumen of the portion of the intestine usually colon existing in a hernial sac is blocked with faeces. This scybalous content of the bowel should be capable of being indented with the finger like putty. Sac and contents are densely adherent to each other. It is always irreducible often obstructed but may not be strangulated.

Classification According to Contents

Omentocoele—omentum.

Enterocoele—intestine.

Cystocoele—urinary bladder.

Litter's hernia—Meckel's diverticulum.

Maydl's hernia.

Sliding hernia.

Richter's hernia—part of the bowel wall.

Precipitating Causes for Inguinal Hernia

Smoking; obesity; respiratory causes like bronchial asthma, tuberculosis, bronchitis; ascites; previous surgery like appendicectomy (injury to ilioinguinal nerve) which causes direct inguinal hernia; chronic constipation due to anorectal strictures; rectal stricture may be due to chronic proctitis (amoebic), tuberculosis of anorectum, previous anorectal surgery, rectal carcinoma or stricture due to lymphogranuloma venereum; urinary problems like benign prostatic hyperplasia (BPH), urethral stricture; straining; multiple pregnancies.

Conservative treatment:

1. **Taxis:** : Patient lying in supine position, contents is pushed with one hand directing with other hand, after flexing hip and knee, and rotating hip internally. It is not done in obstructed hernia, Maydl's hernia, femoral hernia and strangulated hernia.
2. **TRUSS:** Rat-tailed sprung truss is used. Measurement is taken from the tip of greater trochanter to third piece of sacrum. Complications are discomfort, ulceration, strangulation, inflammation.

It may be used in old people who are not fit for anaesthesia and surgery (**Figs 17.35 and 17.36**).

Conservative treatment should be avoided in hernia as much as possible.



Fig. 17.33: Parts of hernia—neck, body and fundus.

Recurrent Hernia

Causes: Infection—most common—50%; haematoma in the wound; early straining; retained indirect sac, after repair of a direct sac (*Pantaloon hernia*); smoking, constipation, obstructive uropathy, old age, nutritional deficiencies; altered tension in repair site; altered collagen synthesis.



Fig. 17.34: Common and rare sites of hernia.

Recurrence rate after—

Bassini's repair—10%
Shouldice repair—1%
Hernioplasty—1 to 3%
Other methods—1 to 5%

Types of recurrent hernias: True or false recurrence—based on type of recurrence—whether inguinal recurrence after inguinal hernia repair (true)/femoral hernia or obturator or other rare types after inguinal hernia repair (false). But presently hernia is classified grossly as groin hernias and so all recurrences are true recurrences. **Clinical features:** Swelling, expansile impulse on coughing, visible scar, reducibility (**Fig. 17.37**). Examination is like inguinal hernia. It is often difficult to categorise it as direct or indirect as earlier surgery has destroyed the true anatomy of the groin. Often it is classified as medial recurrence or lateral recurrence. Incidence of re-recurrent hernia after hernioplasty is 1%.

Incisional Hernia

Incisional hernia is a hernia occurring through a weak scar. Writing case sheets, taking detailed history is similar to inguinal hernia.



Fig. 17.35: Hernia truss. Note the position where sac is supported. It is not commonly used now as it may precipitate strangulation.

Additional history to be collected in history of present illness—Details of surgery patient has undergone earlier. After how long incisional hernia has occurred? History of wound infection, wound dehiscence, whether surgery done was an emergency or elective, and whether tension sutures was placed or not. History of pain, irreducibility and details of precipitating factors has to be asked. Other precipitating factors similar to inguinal hernia like smoking, urinary/respiratory/abdominal symptoms should be asked (**Fig. 17.38**).



Fig. 17.36: Left sided complete inguinal hernia in a patient with Benign Prostatic Hyperplasia (BPH) who is on Foley's catheter. He needs Trans Urethral Resection of Prostate (TURP) with hernioplasty.

Local Examination (Abdomen)

Inspection

Scar, its extent and location, whether healed primarily or secondarily, skin over the scar and swelling is noted. Details of the swelling with expansile impulse on coughing and examination both in lying down and standing are done (**Figs 17.39A and B**).



Fig. 17.37: Recurrent hernia left sided. Note the scar of earlier surgery.

Palpation

Palpation is like for inguinal hernia. Size, extent, impulse on coughing must be confirmed; scar and skin should be palpated. The defect in the abdominal wall must be assessed. It is done after reducing the hernial content with patient in lying down position. Fingers are placed *horizontally* over the hernial defect and patient is asked to raise the head with arms folded over the chest (to contract the abdominal wall muscles) so that the defect is felt clearly. Its size, extent can be assessed well. Assessment can also be done by raising the legs instead of head (**Figs 17.40 and 17.41**).

Gap cannot be assessed in an irreducible hernia.



Fig. 17.38: Lower abdominal incisional hernia adherent to skin.

Factors Responsible for Development of Incisional Hernia

Vertical incision has got higher chances of incisional hernia than horizontal incision; Layered closure of the abdomen has got higher chance than single layer; Continuous closure has got higher chances than interrupted closure; Using of absorbable suture material has got higher chances of hernia than use of non-absorbable sutures; Emergency surgical wound has higher chances than elective surgical wound; Laparotomy for peritonitis, acute abdomen, and trauma



Figs 17.39A and B: Large incisional hernia in standing and lying down position.

can commonly cause incisional hernia; Drainage through the main laparotomy wound may precipitate formation of incisional hernia; Chronic cough, smoking, obstructive uropathy, constipation can precipitate incisional hernia; Diabetes, old age, malnutrition, malignancy, anaemia, hypoproteinaemia, jaundice, ascites, liver disease, uraemia, steroid therapy, immunosuppressive diseases are other precipitating factors (**Fig. 17.42**).

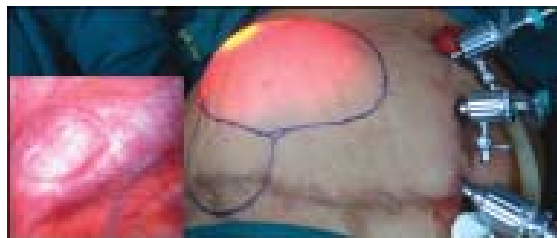


Fig. 17.40: Defect is assessed by placing fingers horizontally in incisional hernia.

Type of defects in incisional hernia

- Small defect
- Large and wide defect
- Very large defect
- Massive/diffuse
- Multiple defects

Note: Size of the defect is important to decide the type of surgical closure in incisional hernia. Midline hernia expels the content more outwards due to contraction of rectus muscles on both sides.

Preoperative Preparations for Incisional Hernia Surgery

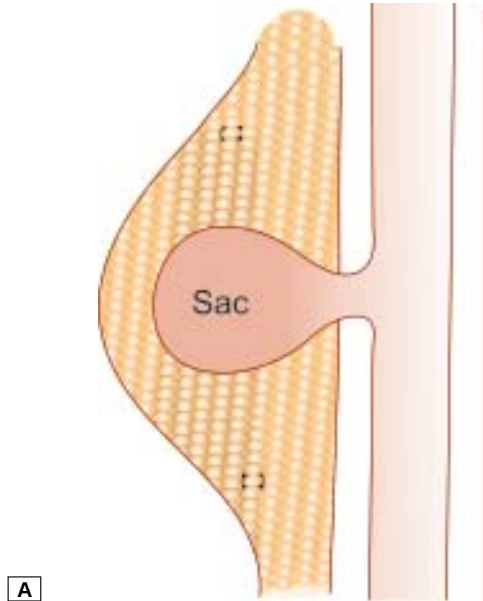
Reduction in weight and control of obesity; Nutritional supplementation, control of anaemia; Treatment for diabetes, hypertension, cardiac diseases, respiratory problems; Treating the precipitating causes; Chest X-ray, US abdomen to be done; Massive incisional hernia after reduction might cause IVC compression, paralytic ileus and diaphragmatic elevation with respiratory embarrassment (*abdominal compartment syndrome*). It is prevented by prior increasing the capacity of peritoneal cavity by creating the pneumoperitoneum using CO₂ so as to increase the peritoneal pressure by 12-15 cm of H₂O, daily for 3-6 weeks. Later definitive surgery is done. Lordosis and back pain may be presenting features. Sac and contents may get adherent to the thin skin over the summit of the hernia leading to skin ulceration and occasionally fistula formation. Often might need resection of the adherent bowel segment.

Epigastric Hernia

It is fatty hernia of linea alba; initially it is sacless (protrusion of extraperitoneal fat) but later develops true epigastric hernia with sac containing contents. It occurs through decussation of the linea alba above the umbilicus. It is 10% common; 20% are multiple *Swiss cheese* pattern. It is often symptomless but later can cause pain, obstruction and strangulation. It is better seen and palpated in *standing position* as a firm nodule which is relatively non-mobile. Abdominal wall lipoma which mimics epigastric hernia is freely mobile. Often it is associated with peptic ulcer and so pain may be due to peptic ulcer, hence gastroscopy should be done in doubtful patients (**Figs 17.43A to C**).

Paraumbilical Hernia

It is midline herniation above or below the umbilicus through a defect *adjacent* to umbilicus. It is common above the umbilicus. It often attains large size and sags downwards. Neck may be narrow with omentum/



A



B



C

Fig. 17.41: Incisional hernia showing visible intestine under thinned out skin.



Fig. 17.42: Laparoscopic view of incisional hernia.

small bowel as contents. Obstruction/strangulation tend to occur. It is commonly associated with obesity and multiple pregnancies. It is common in females; obese; middle or old aged. Swelling, impulse on coughing, dragging pain and reducibility are usual presentations. After reduction firm ring-like fibrous edge is felt (**Fig. 17.44**).

Umbilical Hernia

It is herniation through a weak umbilical cicatrix. It is common in infants and children. It is common in Negroes. It is hemispherical in shape with defect felt during crying. Commonest content is small intestine. It can cause obstruction and strangulation. 95% of umbilical hernias disappear in 2 years. If it persists beyond 2 years, and if the defect is more than 2 cm in size or if associated with complications surgery is indicated. It is operated through an infraumbilical incision; defect is closed with interrupted sutures after ligating the sac. Acquired umbilical hernia occurs in adult life but it is very rare (**Figs 17.45 and 17.46A and B**).

Ventral Hernia

Any protrusion through abdominal wall with the exception of hernia through the inguinofemoral region is defined as ventral hernia. Incisional hernia (80%)



Figs 17.43A to C: Epigastric hernia.

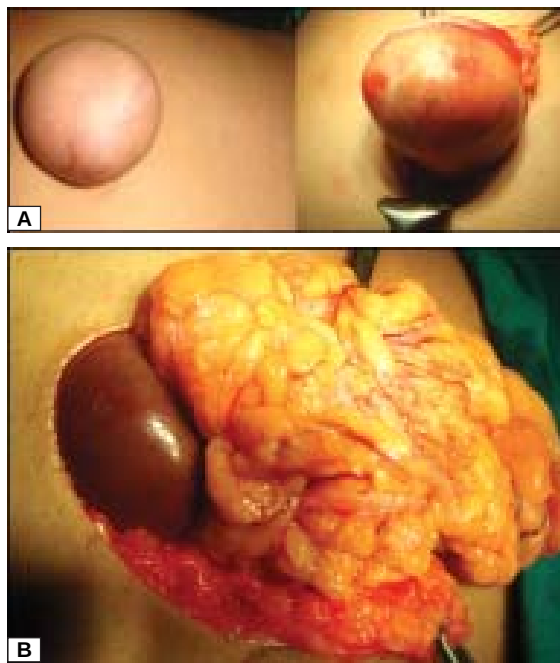


Fig. 17.44: Paraumbilical hernia.



Fig. 17.45: Umbilical hernia

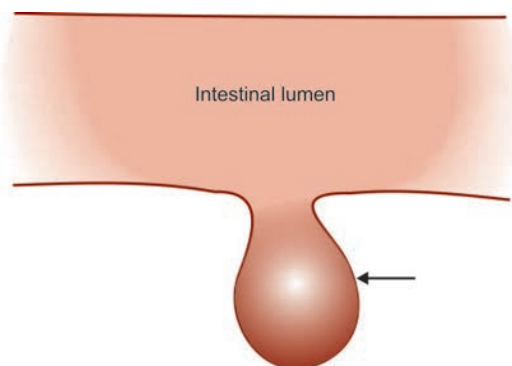
and primary defects in abdominal fascia which can cause umbilical hernia, epigastric hernia, paraumbilical hernia or Spigelian hernia are grouped under ventral hernia. *Ventral hernia can be* – Reducible; irreducible; obstructed; strangulated; single; multiple small defects (*Swiss cheese hernia*) (**Fig. 17.47**). *Causes:* Congenital defect; obesity; smoking; chronic cough.

Richter's Hernia

It is herniation of a portion of circumference of intestine usually small bowel leading into gangrenous change. But patient presents with features mimicking gastroenteritis without any signs of intestinal obstruction. Eventually it leads to perforation and peritonitis. It is common in femoral hernia. It is treated by resection and anastomosis and repair (**Figs 17.48 and 17.49**).

Sliding Hernia

Posterior wall of the sac is formed by parietal peritoneum and also by sigmoid colon/caecum/urinary bladder. It occurs exclusively in males and common



Figs 17.46A and B: Umbilical hernia strangulated (irreducible, tense, tender) showing also gangrenous bowel on table.



Fig. 17.47: Large ventral hernias.

on left side. It attains large size and its content is usually small bowel. Posterior wall should not be separated from the sac. Sac is excised only partially and then is pushed into peritoneal cavity. Mesh repair is done afterwards (**Fig. 17.50**).

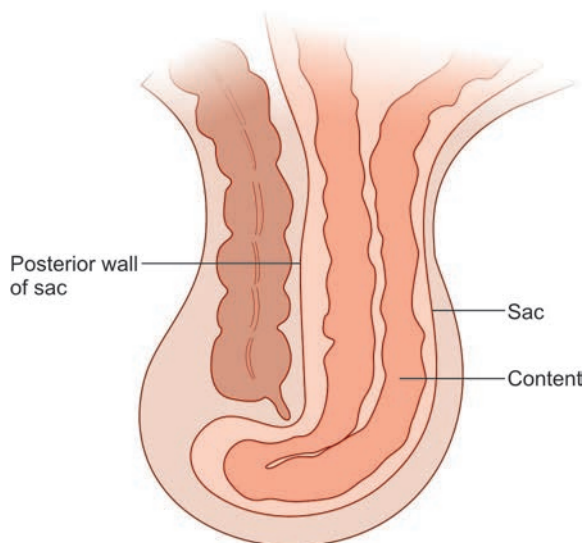


Fig. 17.48: Richter's hernia.

Pantaloon Hernia

Inguinal hernia containing both direct and indirect sacs is called as *pantaloon hernia* but it presents as direct hernia. It is also called as *double hernia*, *saddle hernia* or *Romberg hernia*. So in all cases of direct hernia indirect sac should be looked for. Condition is one of the causes for recurrence.

Strangulated Hernia

It is due to compromised blood supply of the contents of the hernia like bowel/omentum causing toxicity, tenderness at the site. There is no impulse on coughing, and is irreducible and tense. Features of intestinal obstruction are present if the content is bowel. Narrow neck and adhesions are the causes of strangulation. It is treated by emergency surgery.

Clinical features of strangulated hernia: Presents with sudden severe pain, initially over a pre-existing hernia which later becomes generalised over the abdomen; persistent vomiting, constipation and distension of the abdomen; *hernia is tense, severely tender, irreducible, without any expansile impulse on coughing. Rebound tenderness is diagnostic.* Features of toxicity and dehydration; electrolyte imbalance; abdominal distension with guarding and rigidity oliguria are other features.

Strangulation during infancy: Incidence is 4%. Female to male ratio is 5:1. In female infants, the content may be ovary with or without fallopian tube. Taxis may be dangerous as during taxis, contusion and rupture of the intestinal wall can occur. Reduction en masse may mask the gangrenous bowel existing in the sac. Inner gangrenous loop of Maydl's hernia may be missed. Rupture of the sac extraperitoneally can also be the possibility. **Taxis have no role in femoral hernia and strangulated hernia.** If tried, contusion, reduction en masse and rupture of the sac can occur. **In strangulated omentum** features of obstruction are not present. Omentum becomes congested, oedematous and black in colour which secretes toxic fluid with secondary bacterial infection. But here, the sepsis is slower initially than that of strangulated intestinal obstruction. Eventually the infection spreads causing diffuse peritonitis. Downward spread of infection can cause scrotal abscess (**Figs 17.51 to 17.53**).

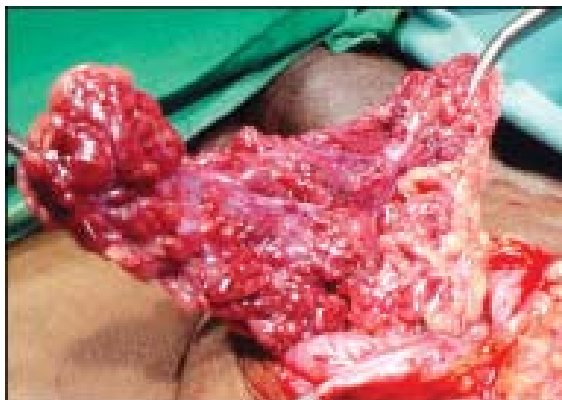


Fig. 17.49: Richter's hernia with gangrene of part of the circumference of the bowel with perforation in a case of femoral hernia.



Fig. 17.50: Sliding hernia.

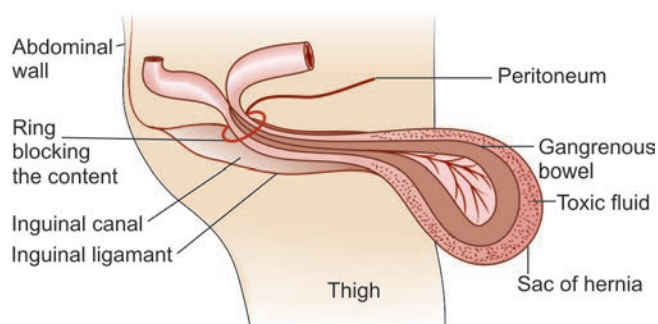
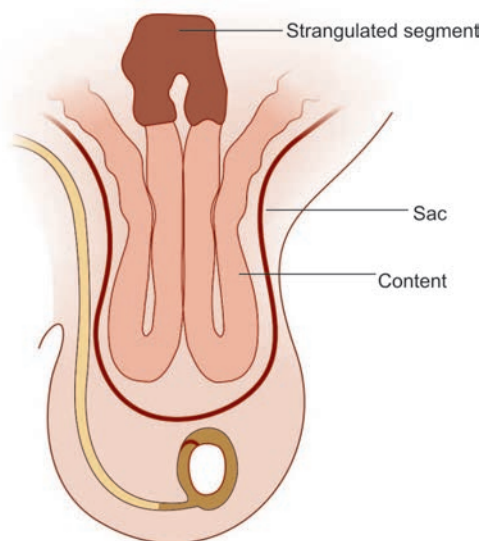


Fig. 17.51: In strangulated hernia if the content is strangulated omentum, then omentum is excised and repair is done. Mesh is usually not used in strangulated hernia.



Figs 17.52A and B: Strangulated enterocele with irreducibility, absence of impulse on coughing, signs of acute inflammation, tense and tender mass with features of intestinal obstruction. Bowel strangulation is obvious during surgery. Here bowel loop in the form of 'W' lies in the hernial sac and center of the portion of the W is strangulated. It may get reduced 'en-masse'. Strangulation of center part is common (**Fig. 17.54**).

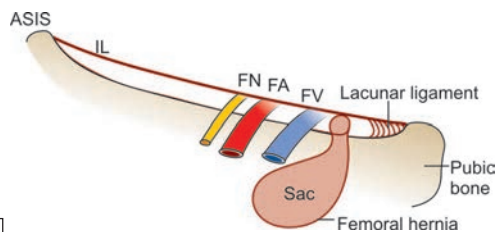
Femoral Hernia

It is common in females. It occurs in medial most part of the femoral canal.

Surgical Anatomy of Femoral Canal

It is the most medial compartment of the femoral sheath which extends from femoral ring above, to saphenous opening below. It contains fat, lymphatics, lymph node

of Cloquet. It is 1.25 cm long and 1.25 cm wide at the base. Below it is closed by cribriform fascia. Femoral ring is bounded anteriorly by inguinal ligament; posteriorly by iliopectineal ligament of Cooper, pubic bone and fascia covering the pectineus muscle; medially by concave, sharp lacunar (Gimbernat's) ligament; laterally by a thin septum separating from femoral vein (**Figs 17.55A and B and 17.56**).



A



B

Fig. 17.53: Diagram showing the strangulated hernia with toxic fluid and site of obstruction.

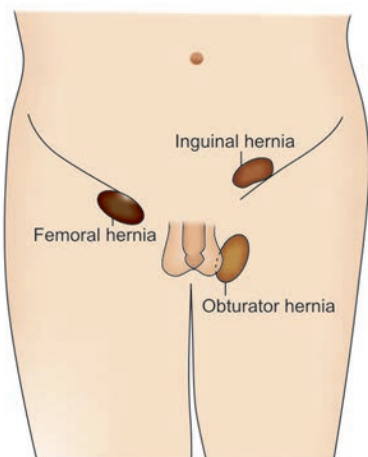
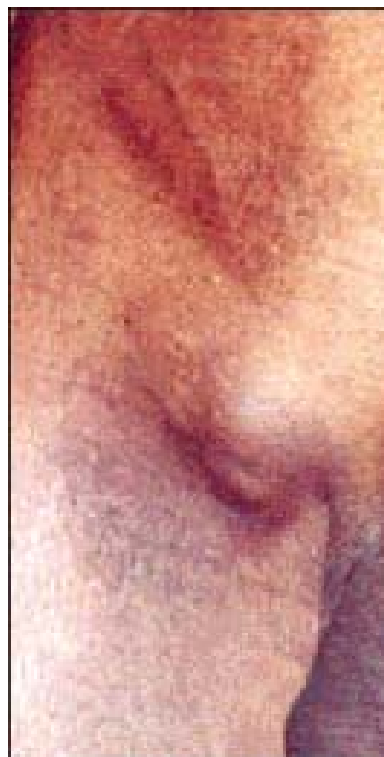


Fig. 17.54: Maydl's 'W' hernia.

Pathology in femoral hernia: Through femoral canal, hernial sac descends down vertically upto saphenous opening and then escapes out into the loose areolar tissue to expand out like a *retort*. Because of its irregular pathway and narrow neck, it is more prone for obstruction and strangulation. During surgery, precaution should be taken about the femoral vein and pubic branch of obturator artery (or accessory obturator artery) which often may get injured leading to torrential haemorrhage.

Clinical features: Common in females (2:1), common in multiparae; Rare before puberty; 20% occurs bilateral, however more common on right side; presents as a swelling in the groin *below and lateral* to the pubic tubercle (Inguinal hernia is *above and medial* to the pubic tubercle); swelling, impulse on coughing, reducibility, gurgling sound during reduction, dragging pain, are the usual features. When obstruction and strangulation occurs which is more common (due to rigid opening of femoral canal), presents with features of obstruction—pain, tender, inflamed, irreducible swelling without any impulse. They also present with abdominal distension, vomiting, and feature of toxicity. Often femoral hernia can be associated with inguinal hernia also. *Gaur* (Surgeon Bombay Hospital, India)



Figs 17.55A and B: Femoral hernia is common in females. It occurs below and lateral to pubic tubercle. Herniation occurs through femoral ring—medial most part of the femoral canal. ASIS—Anterior Superior Iliac Spine; IL—Inguinal Ligament; FN—Femoral Nerve; FA—Femoral Artery; FV—Femoral Vein.

dilatation of one or both veins (**Fig. 17.57**). *Remember—presentations may be* – No lump/expansile impulse on coughing in saphenous opening/visible impulse which get reduced and more prominent on coughing/irreducible tender, tense swelling in femoral region often extend into inguinal region from below upwards.

In congenital dislocation of hip, femoral hernia occurs *behind* the femoral vessels—*Narath's femoral hernia*. If sac lies under the pectineal fascia, is called as *Cloquet's hernia*. *Strangulation and Richter's hernia are common in femoral hernia*. Often on medial side, a portion of bladder forms the wall of the femoral hernial sac—*sliding femoral hernia*.

Differential Diagnosis for Femoral Hernia

Inguinal hernia: It is confirmed by its location above and medial to pubic tubercle and invagination test is positive and full whereas in femoral hernia invagination

test is negative and empty. Impulse will be felt in saphenous opening and Zieman's test will demonstrate impulse in ring finger.

Prevascular type of femoral hernia: It is in front of femoral vessels. It bulges just underneath the inguinal ligament; shows wider neck; impulse on coughing is easily felt; easily reducible; strangulation is rare; difficult to repair.

Saphena varix: It is saccular dilatation of the great saphenous vein near saphenofemoral junction. It apparently shows impulse on coughing which is actually tremor imparted on the feeling fingers like a jet of water entering and filling the pouch – *Cruveilhier's sign of saphena varix*. There will be associated long saphenous vein varicosity. Blue discolouration of the skin over it is often obvious. Saphena varix is softer.

Enlarged Cloquet's inguinal lymph node: It mimics irreducible femoral hernia. One has to look for focus like infection or neoplastic.

Psoas abscess: It is usually a cold abscess due to tuberculosis of thoracolumbar spine (Pott's disease). It is painless reducible swelling below the inguinal ligament lateral to the femoral artery pulsation; often there is swelling above in iliac region which is then cross fluctuant across inguinal ligament. Spine should be examined. US abdomen and groin confirms pus. CT pelvis is confirmative.

Enlarged psoas bursa: Psoas bursa gets enlarged in osteoarthritis of hip joint as this bursa which is located in front of the hip joint and behind psoas major communicates with joint cavity. Clinically it produces a tense, cystic swelling in front of the hip joint below the inguinal ligament which diminishes in size when joint is flexed.

Femoral aneurysm: It is pulsatile (expansile), mobile only side-to-side; compressible swelling. But if thrombosed, pulsation may not be evident and mimic irreducible femoral hernia.

Hydrocele of femoral hernial sac: It presents as soft, fluctuant swelling which is transilluminant.

Ectopic testis: When ectopic testis is located in femoral triangle it mimics femoral hernia. Empty scrotum,

absence of impulse, firm swelling with testicular sensation on pressure is typical.

Lipoma in femoral region: It is soft, freely mobile, smooth, lobulated with positive slip sign.

Femoral hernia—medial to femoral vein.
 Narath's hernia—in front of femoral artery.
 Hesselbach's hernia—lateral to femoral artery.
 Cloquet's hernia—behind femoral vessels.
 Laugier's hernia—through lacunar ligament.

Rare Hernias

Obturator hernia is herniation through obturator canal. Mainly presents as intestinal obstruction; often pain in knee joint (referred); rarely as a swelling in the medial side of the thigh (**Fig. 17.58**).

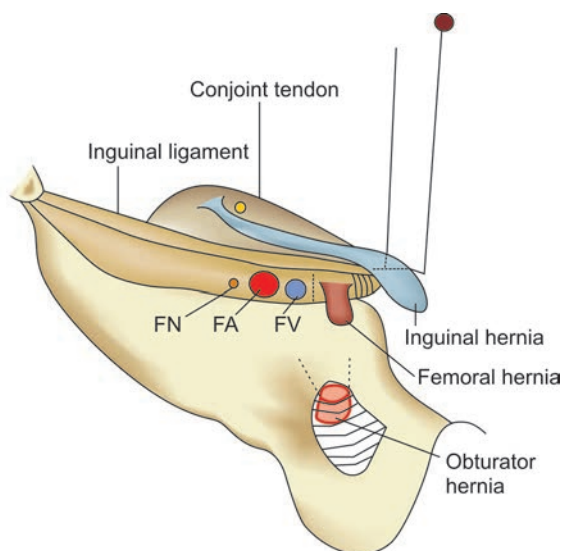


Fig. 17.56: Anatomical locations of femoral and inguinal hernia. Inguinal hernia is above and medial to pubic tubercle. Femoral hernia is below and lateral to pubic tubercle. Also note the location of the obturator hernia below in Scarpa's triangle.

Funicular hernia: It is prevesical direct hernia which is prone for strangulation, is herniation of prevesical fat, bladder, or intestine through a small defect in the medial part of the *conjoint tendon* just above the pubic tubercle.

Spigelian hernia: It is an interparietal hernia occurring at the level of arcuate line through spigelian point with sac lying deep to internal oblique or between internal oblique or external oblique muscles (**Fig. 17.59**).

Lumbar hernia: It is herniation through either superior lumbar triangle bounded by sacrospinalis, 12th rib, posterior border of internal oblique—(*Grynfeltt-Lesshaft's triangle*) or inferior lumbar triangle bounded by latissimus dorsi, external oblique and iliac crest (*Petit's triangle*). It is common through inferior triangle. It should be differentiated from lipoma, cold abscess, lumbar phantom hernia.

Phantom hernia is a muscular bulge as a result of local muscular paralysis due to interference with nerve supply of the affected muscles like in poliomyelitis. It is common in lumbar region. It is often seen in lower abdomen.



Fig. 17.57: *Gaur sign* (dilated superficial epigastric vein) in a patient with femoral hernia (Gaur, Mumbai, India).

Infantile hernia: It is clinically difficult to diagnose; often it is diagnosed on table. Processus vaginalis is closed at internal ring and hernial sac either invaginates processus vaginalis as 'inverted umbrella' or comes behind processus vaginalis.

18

Examination of Inguinoscrotal and Scrotal Swelling

In this chapter inguinoscrotal and scrotal swellings other than inguinal and femoral hernias are discussed. Hydrocele and other differential diagnosis of groin swellings are discussed here.

History taking begins with:

Name:

Age:

Address:

Occupation:

Funiculitis occurs in young age. Testicular tumour occurs in early adult age (seminoma) but can occur in young (teratoma). Varicocele presents in adolescents and young. Filarial funiculitis, orchitis, lymph varix are common in Orissa, West Bengal, coastal regions. Carcinoma of scrotal skin is seen in elderly. Torsion testis is seen in adolescents. Hydrocele is common in adult. Epididymal cyst, spermatocele are seen in adult. Tuberculous orchitis is seen in young individuals.

Carcinoma of scrotal skin is often occupation related those who come in contact with soot (*Chimney sweep's*

cancer); tar or oil (*Mule spinner's disease*). Prolonged standing may cause varicocele.

History

History of Present Illness

Swelling: Like any other swelling detailed history should be asked – mode of onset, progress, regress, history of trauma, whether swelling disappears on lying down (reducible hernia will disappear on lying down, hydrocele will not disappear). Rapid increase in size occurs in testicular tumours. Trauma may precipitate haematocele or hydrocele. Sudden pain and swelling in scrotum is probably due to acute epididymo-orchitis or pyocele. Inguinal hernia begins in groin; hydrocele, testicular tumour begins in scrotum; varicocele begins in root of the scrotum. Lipoma of cord, encysted hydrocele of the cord appears in groin or near root of the scrotum. Undescended testis presents with swelling in the groin with empty scrotum. In bilateral swellings the side on which it appeared first and after

Inguinoscrotal and scrotal swellings

Encysted hydrocele of the cord
Varicocele
Lymph varix
Funiculitis
Diffuse lipoma of the cord
Ectopic testis
Undescended testis
Retractile testis
Torsion testis
Testicular tumour
Inguinal or iliac lymphadenopathy
Abscess in the groin – inguinal region
External iliac artery aneurysm
Scrotal oedema
Epididymal cyst
Spermatocele
Extravasation of urine

Femoral swellings

Inguinal lymph nodes
Saphena varix
Psoas abscess
Psoas bursa enlargement
Lipoma in femoral triangle
Femoral artery aneurysm
Hydrocele of femoral hernia
Ectopic testis

how long has it appeared on opposite side should be asked. Hydrocele can be bilateral; varicocele is more common on left side but can be bilateral; bilateral impalpable undescended testes are called as *cryptorchidism*. Other swellings in epigastric region in the abdomen (due to palpable enlarged paraaortic lymph nodes)/in the supraclavicular region due to enlarged lymph nodes may be elicited in history often. Scrotal oedema may be due to filariasis or part of generalised anasarca. Trauma on bulb of urethra may cause urinary extravasation and swelling in the scrotum. Gonococcal urethritis with stricture also can cause extravasation and scrotal swelling – *watering can perineum*.

Swelling with heaviness in the scrotum may be initial presentation of testicular tumour.

Pain: Epididymo-orchitis, funiculitis presents with pain and often fever. History of severity of pain, onset, progress, whether initially painless later became painful (in testicular tumour) should be asked. Haematocoele, pyocoele can be severely painful. Torsion testis presents with pain often precipitated by straining due to sudden violent contraction of the cremaster. Severe pain in scrotum and groin with fever, redness and swelling may be features of acute epididymo-orchitis. Periodic mild fever with pain, discomfort and swelling in the scrotum and spermatic cord are the features of filarial epididymo-orchitis.

Presence of fever: Fever often with chills should be asked for. Fever is due to infection, filarial orchitis, pyocoele, etc. Evening rise of temperature may be feature of tuberculous epididymitis or funiculitis. History of cough and haemoptysis suggests associated pulmonary tuberculosis.

Trauma: History of trauma is important as scrotum being entirely outside is prone for it. Haematocoele, urinary extravasation in urethral injury, haematoma scrotum in perineal injury – are important conditions to be remembered. Existing disease may be obvious after trauma.

Infertility: It may be the presenting complaint in varicocele or cryptorchidism.

Past History

History of tuberculosis; old trauma; sexual exposure (in gonococcal urethritis or syphilitic orchitis) are earlier important signs should be asked for. Patient might

have undergone surgery for inguinal hernia (post-herniorrhaphy hydrocele) or hydrocele surgery.

Personal History

History of sexual contact earlier should be elicited.

Swellings which reduce on lying down in the groin

Reducible inguinal hernia – reduces rapidly
Varicocele – reduces spontaneously with elevation of scrotum
Lymph varix reduces slowly.

General Examination

Pallor, nutrition, oedema, jaundice should be checked; pulse and blood pressure should be recorded. Anaemia and malnutrition may be features in tuberculosis or advanced malignancy.

Local Examination

First always patient should be examined in *standing position* later in lying down position.

Inspection

Swelling: Swelling in the scrotum may be due to hydrocele, epididymal cyst, spermatocele, scrotal oedema, and varicocele. Hydrocele causes obvious swelling often very large. In encysted hydrocele of the cord, lipoma of the cord swelling may be in the groin or root of the scrotum. Swelling in the groin or superficial inguinal pouch may be due to undescended testis or ectopic testis (**Figs 18.1A and B**). Here empty scrotum is obvious on inspection. Scrotal oedema may be associated with penile oedema. It may be due to scrotal cellulitis, cardiac/renal/hepatic causes, filariasis, advanced secondaries in the inguinal lymph nodes blocking cutaneous lymphatic drainage, and extravasation of urine. Extravasation may be due to urethritis or urethral trauma. Penis may be buried in large hydrocele, large inguinoscrotal hernia, and scrotal oedema.

Skin over the scrotum/swelling/groin should be inspected. It is red and oedematous in funiculitis, and orchitis. Acutely inflamed skin with redness is also often a feature of torsion testis (**Fig. 18.2**). Strangulated hernia also shows signs of inflammation over the skin.



Figs 18.1A and B: Undescended testis in two patients. (A) Bilateral and testes are not present even in groin—Cryptorchidism. Note the underdeveloped empty scrotum. (B) It is unilateral (right sided) with visible testis (swelling) in the inguinal region.

Its extent is from scrotum to groin. Skin over the swelling may be stretched with *loss of rugosity* in long standing hydrocele. Rugosity will also be lost in syphilitic orchitis, tuberculous epididymitis and testicular tumours. Skin may be stretched often in scrotal oedema. Filarial scrotal oedema is non-pitting whereas scrotal oedema due to other causes is pitting in nature. One has to remember that common cause of hydrocele is filarial. Clinician should be able to



Fig. 18.2: Typical look of torsion testis – right sided.

differentiate hydrocele from scrotal oedema. Ulcer in the scrotal skin may be due to **scrotal carcinoma** which will be having raised and everted edge, slough in the floor. All features of an ulcer explained in Chapter 2: ‘Examination of an Ulcer’ should be mentioned. **Testicular tumour** occasionally can fungate through scrotal skin (can be anywhere but *usually anterolateral*) and present as an ulcer. Syphilitic **gummatous ulcer** is located always *on front (anterior)* of the scrotum which is adherent to testis due to syphilitic orchitis. It is punched out with wash leather slough. **Tuberculous epididymitis** causes ulceration on the *posterior aspect* of the scrotum with an undermined edge. Only in anteverted testis positions are reversed. Testis may protrude out as a granulating mass in severe infection called as **hernia testis**. Hydrocele fluid may protrude out of the tunica vaginalis testis through dartos as **hernia of the hydrocele**. Scrotal skin gangrene is a feature of **Fournier’s gangrene**. Multiple discharging sinuses in the scrotal skin are the features of the gonococcal urethritis with discharging urine—**watering can perineum**. **Multiple sebaceous cysts** are common in scrotum. Scrotum may show whitish vesicle containing lymph due to filariasis (**lymph scrotum**) which may rupture causing **lymphorrhagia**.

Sinus in the scrotum can develop due to infection, postoperative cause or tuberculosis. Urinary fistulas in the scrotum can occur (**Fig. 18.3**).

Impulse on coughing: Inguinal hernia shows expansile impulse on coughing. Varicocele and lymph varix also



Fig. 18.3: Discharging sinus in the scrotum.



Fig. 18.4: Left sided varicocele.

show impulse on coughing but with fluid thrill (**Fig. 18.4**).

Groin should be inspected for swelling (inguinal nodes), ulceration, and fungation. Ulceration can occur in groin due to secondaries in the lymph nodes, bubo, tuberculosis, etc.

Palpation

Palpation of the Swelling

Position and extent: Exact location of swelling on inspection is important whether it is in the inguinal/inguinoscrotal/scrotal. Encysted hydrocele of the cord is located usually in the middle of the cord near the root of the scrotum occasionally in the groin. It will not extend proximally above. Lipoma of the cord, funiculitis (inflammation of vas deferens, filarial—common), ectopic testis in superficial inguinal pouch are other groin swellings to be considered. Skin should be held to see the fixity (**Fig. 18.5**).



Fig. 18.5: Skin should be held to find out the skin fixation.

Get above the swelling: In standing position cord is palpated for structures by placing thumb in front and fingers behind the root of the scrotum. In hydrocele one can get above the swelling – means only cord structures are felt and nothing else. In inguinoscrotal hernia, one cannot get above the swelling. Cord with additional structures are also felt. This is important test to confirm scrotal swelling (**Figs 18.6A and B**).

Reducibility: Inguinal and inguinoscrotal hernia is reducible. By taxis hernial contents are gently reduced and emptied into the abdominal cavity. Hernia gets reduced abruptly and rapidly. Hydrocele is not reducible. Exception is congenital hydrocele which commu-



Figs 18.6A and B: (A) One can get above the swelling in hydrocele. (B) In inguinoscrotal swelling getting above the swelling is not possible.

nicates with abdominal cavity. Congenital hydrocele is usually associated with tuberculous ascites. Varicocele and lymph varix also gets reduced while lying down but slowly and gradually.

Impulse in coughing: Hernia shows expansile impulse on coughing. Varicocele and lymph varix also show impulse on coughing like a fluid thrill but it is not expansile.

Like any other swelling, **size, shape, surface, warmth, tenderness, consistency** should be checked. Encysted hydrocele is fluctuant, cystic (Paget's test

is positive), transilluminant. Lymph varix is soft, cystic and doughy. Varicocele is soft, with typical feel of 'bag of worms'. Hydrocele is smooth and soft (firm if it is tensely cystic).

Fluctuation: This is essential test for hydrocele. Upper part of the scrotum is held between thumb and fingers of one hand to *steady* the swelling; thumb and fingers of other hand are held at lower pole. Intermittent pressure from lower fingers will push apart the fingers over upper part and vice versa. Test is repeated in opposite direction. It is important to elicit fluctuation in two directions. It is also important to fix the swelling prior to eliciting the fluctuation (**Figs 18.7A and B**).



Figs 18.7A and B: Fluctuation should be elicited in two directions in hydrocele after fixing the swelling using fingers.

In a small swelling Paget's test is used. In bilocular hydrocele, i.e. swelling in the groin and hydrocele with band-like narrowing near external ring, fluctuation can be elicited across external ring above and below – *cross fluctuation* (Other swellings which are cross fluctuant are—psoas abscess, ranula, and compound palmar ganglion). Hydrocele, encysted hydrocele, epididymal cyst, spermatocele, abscess are fluctuant swellings in the scrotum.

Transillumination/translucency: It is done in a dark room using a pen torch. Pen torch is placed laterally in the anterior part of the scrotum. Never place it posteriorly as testis will interfere with proper illumination. Red glow of translucency is seen in the scrotum which is better appreciated using roll of thick paper or X-ray sheet placed on the opposite side (medially) especially with day light. Hydrocele becomes non-transilluminant due to thick dartos, thick unclear fluid, thick sac, haematocele, chylocele, pyocele. In epididymal cyst, it is brilliantly transilluminant (**Figs 18.8A and B**).



Figs 18.8A and B: Hydrocele which is transilluminant.

Traction test: It is the test for encysted hydrocele of the cord. Swelling is located above the testis which is mobile but becomes immobile once testis is pulled down from the swelling. It is used to differentiate it from epididymal cyst.

Palpation of Testis

Position: Testis may be in normal position with testis in front, epididymis behind and globus major upwards. In *anteverted testis*, epididymis lies anteriorly and body posteriorly. In *inverted testis*, testis lies upside down with globus major inferiorly. In *incompletely inverted testis*, testis lies horizontally. Inverted or incompletely inverted testis precipitates torsion testis. Often these changes are bilateral.

Size: Normal size is 3.75 cm above downwards; 2.5 cm from anterior to posterior; 1.8 cm side-to-side. It weighs 10-15 gram. Atrophied testis is smaller in size. It may be due to undescended testis or due to earlier mumps attack or developmental defect. Atrophy may be unilateral or bilateral. Larger testis may be due to tumour or filarial orchitis or syphilitic gumma. *Weight of the organ* in relation to size should be assessed. It is done by balancing the testis on the palm of the hand. Testis is *heavier in testicular tumour and haematocele*. It is lighter in gummatous testis even though size is large (**Fig. 18.9**).

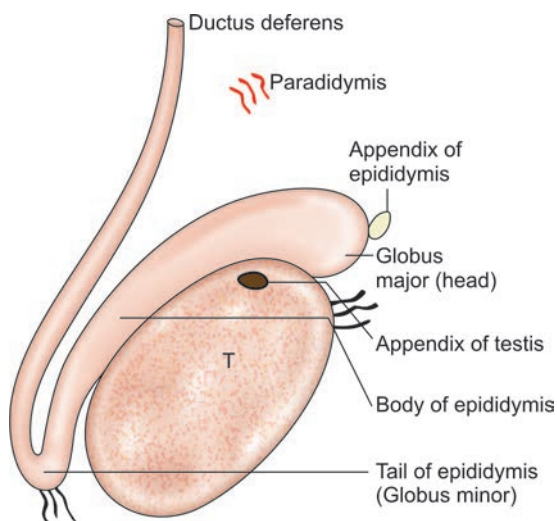


Fig. 18.9: Anatomy of testis.

Testicular sensation: It is sickening sensation / pain felt in the abdomen (at the level of umbilicus – T10) by the patient when a mild pressure is applied (gentle squeezing) over the testis. It is absent in testicular tumour, syphilis (Gumma), leprosy, chronic haematocoele. Both side testes should be palpated. True testicular pain is located in the lower abdomen at the level of internal ring in accordance with Brown's law. In suspected malignancy of testis, it should be avoided or gentle to prevent possible spread by squeezing into veins and lymphatics.

Palpation of Epididymis

It is firm uniform structure along the posterior aspect of the testis with upper head - *globus major*, middle body, lower tail - *globus minor*. **Tuberculosis** commonly involves epididymis mainly *globus minor* (tail) initially (due to retrograde spread along vas deferens). Head is involved by haematogenous spread. Epididymis tail is thickened, nodular and often tender. Later when entire epididymis is involved, epididymis will be enlarged, *craggy*, firm. Eventually coagulation necrosis → softening → cold abscess occurs on the posterior aspect of the scrotum → sinus/ulcer formation behind. Firm, irregular enlarged epididymis is common in filariasis. **Acute epididymo-orchitis** is smooth, soft tender swelling posteriorly due to bacterial or viral (mumps) causes. After prostatectomy retrograde bacterial infection can cause acute infection.

Palpation of Spermatic Cord

Cord is palpated for vas deferens. Vas deferens is palpated at the root of the scrotum between thumb and index finger *together on both sides* (Fig. 18.10). Normally it slips between fingers like whipcord. Vas is thickened and tender in epididymo-orchitis—acute or chronic due to funiculitis. It is thickened and beaded in tuberculosis of vas. It is thickened and tender in filariasis. Soft, doughy feeling is felt in lymph varix;



Fig. 18.10: Method of palpation of vas.

like 'bag of worms' in varicocele of spermatic cord. Rarely testicular tumour spreads along the spermatic cord causing it nodular and hard.

Palpation of Lymph Nodes

Scrotal skin drains into inguinal lymph nodes; testis and epididymis drains into pre- and para-aortic lymph nodes at the level of origin of testicular artery – transpyloric nodes. Inguinal and iliac nodes may be enlarged in scrotal skin conditions and also when testicular tumour infiltrates the tunica and scrotal skin. Testicular tumour spreads to para-aortic nodes and then to left sided supraclavicular nodes.

Systemic Examinations

Abdomen Examination

Respiratory system examination is important as secondaries can occur in testicular tumours. In tuberculosis primary focus may be in lungs.

Investigations

- Blood—smear for microfilariae; syphilis; ESR.
- Urine for culture, AFB (early morning specimen).
- Chest X-ray to see lung secondaries from testicular tumour as 'cannon ball' type; to see pulmonary tuberculosis.
- Tumour markers in testicular tumour— α -feto protein (AFP); β HCG.
- US of scrotum to see haematocoele, pyocoele, secondary hydrocele, varicocele, testicular tumour. It is very useful in all scrotal diseases (FNAC is contraindicated in testicular tumour. Scrotal approach is also contraindicated).
- CT abdomen to see nodal metastases and liver secondaries.

Filarial	Epididymo-orchitis—both testis and epididymis involved; thickened tender vas
Tuberculosis	Epididymitis—involving epididymis only; rather late, testis is involved only rarely; beaded vas
Syphilis	Orchitis—only testis is involved; rather late epididymis rarely may get involved; vas deferens not involved

Fournier's Gangrene

It is also called as *idiopathic gangrene of the scrotum*. It is a vascular gangrene of infective origin, caused by Haemolytic streptococci, microaerophilic streptococci, staphylococci, *E. coli*, *Clostridium welchii*. There will be fulminant inflammation of the scrotal skin and subcutaneous tissues resulting in *obliterative arteritis* of the arterioles of the scrotal skin leading into cutaneous gangrene.

Clinical features: Condition is common in old age; presents with sudden pain, redness, blackening in the scrotum, fever, severe toxicity; Very fast spreading cellulitis of scrotal skin occurs extending to the groin and often to anterior abdominal wall; extensive skin sloughing occurs *leaving normal testis exposed*. Sometimes toxicity is so severe that they may go for renal failure and other complications; sometimes the condition may worsen rapidly leading to death. Minor perineal injury, infection of anal fissure, drainage of periurethral abscess may precipitate the condition (**Figs 18.11A and B**).

Hydrocele

It is the collection of fluid between the two layers of tunica vaginalis of the testis.

Types (Fig. 18.12)

1. Congenital.
2. Acquired: (a) Primary and (b) Secondary.

Aetiology: *Defective absorption* of hydrocele fluid by the tunica vaginalis, probably due to damage to the endothelial wall by low grade infection. *Excessive production* of fluid—as in secondary hydrocele; *Interference with drainage* of the fluid by lymphatic vessels of the cord; *Communication into the peritoneal cavity* are other causes. **Hydrocele fluid** is amber colored with specific gravity of 1.022 to 1.024. It contains water, salts, albumin, and fibrinogen. Per se hydrocele fluid does not clot, but gets activated if it comes in contact with the blood, fibrinogen and clots firmly. Often fluid contains cholesterol and tyrosine crystals (**Fig. 18.13**).

Cysts which contains cholesterol crystals (1) Vaginal hydrocele; (2) Branchial cyst; (3) Dentigerous cyst.



Figs 18.11A and B: Fournier's gangrene.

Primary Vaginal Hydrocele

It occurs in middle aged, common in tropical countries. Testis is not palpable, usually attains a large size (unlike secondary hydrocele which are small except in filarial hydrocele). *Fluctuant* (It is elicited by fixing the hydrocele with hand and feeling for the fluid movements using fingers placed in *two perpendicular*

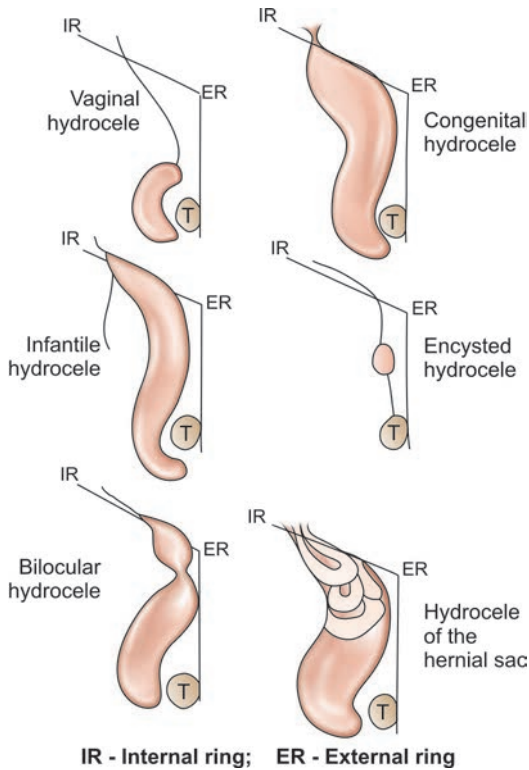


Fig. 18.12: Types of hydrocele.

directions. Note: A relaxed muscle can demonstrate fluctuation in one direction even though there is no fluid in it; initially transilluminant (elicited in front of the swelling side-to-side), but long standing hydrocele is non-transilluminant (due to thickened dartos, thickened spermatic fascia, thickened hydrocele sac, infected content, chylous fluid, often filarial hydrocele, haematocele). One can get above the swelling. Testicular sensation can be elicited in vaginal hydrocele by transmitting the pressure sensation through the fluid (Figs 18.14 to 18.16).

Swellings which are brilliantly transilluminant

1. Vaginal hydrocele
2. Epididymal cyst
3. Cystic hygroma
4. Ranula
5. Meningocele



Fig. 18.13: Hydrocele fluid.



Figs 18.14A and B: Large hydrocele in two patients. Note penis is buried in the scrotum. It is often difficult to differentiate hydrocele from hernia. Get above the swelling and impulse on coughing are the two clinical methods used to differentiate.

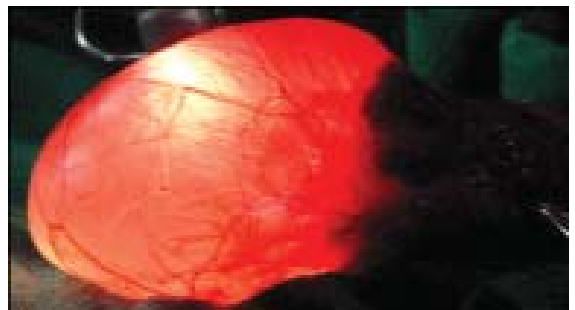


Fig. 18.15: On table transillumination in hydrocele – brilliant transillumination.



Fig. 18.16: Vaginal hydrocele.

Infantile Hydrocele

Here tunica and processus vaginalis (hydrocele) are distended up to internal ring, but sac has no connection with the general peritoneal cavity.

Congenital Hydrocele

Here processus vaginalis communicates with the peritoneal cavity. As this communicating orifice is too small, bowel and/or omentum do not descend and so hernia usually will not develop. While lying down, fluid disappears gradually and while standing fluid recollects. Hydrocele cannot be emptied by digital pressure due to 'inverted ink bottle' effect. Ascites, tuberculous peritonitis are the aetiologies for the same in an adult (**Fig. 18.17**).



Fig. 18.17: Hydrocele in a child. It mimics hernia. It is due to patent processus vaginalis. Treatment is like hernia – herniotomy only through inguinal approach.

Encysted Hydrocele of the Cord

It is a smooth, soft, oval, fluctuant, transilluminant swelling associated with the spermatic cord in inguinal or inguinoscrotal region. Impulse on coughing, reducibility is absent. Testis is felt normal. It is due to persistent patent small portion of the tunica vaginalis in the cord but is closed above and below. On gentle traction to the testis, the swelling becomes less mobile (traction test). **Differential diagnosis:** Epididymal cyst, inguinal hernia.

Hydrocele en Bisac (Bilocular Hydrocele)

Hydrocele has got two intercommunicating sacs, one above and one below the neck of the scrotum. Upper one lies superficial or in the inguinal canal or may insinuate itself in between the muscle layers—*cross fluctuant*. Other condition where cross fluctuation is elicited: (1) Plunging ranula; (2) Compound palmar ganglion; (3) Psoas abscess.

Hydrocele of the Canal of the Nuck

It occurs in females, in relation to the round ligament, always in the inguinal canal.

Hydrocele of the Hernial Sac

It is due to adhesions of the content; fluid secreted will collect in the hernial sac and forms hydrocele of the hernial sac. It occurs in 5% of inguinal hernia cases.

Secondary Hydrocele

Causes: Infection: filariasis; tuberculosis of epididymis; syphilis. Injury: trauma, post-herniorrhaphy hydrocele. Tumour: malignancy. Secondary hydrocele rarely attains large size. It is usually small, lax and testis is usually palpable (unlike primary hydrocele). Exception is secondary hydrocele due to filariasis.

Post-herniorrhaphy hydrocele: It is a secondary hydrocele occurring after the surgery for inguinal hernia. It is due to the damage to lymphatic vessels of the tunica vaginalis and is 0.2% common. It is treated like any hydrocele but usually after about 6 months.

Filarial hydrocele and chylocele: It occurs commonly in coastal region and in and around the Equator. It



Fig. 18.18: Chylocele.

usually occurs after repeated attacks of filarial epididymitis. Hydrocele is usually of large size and the sac is thickened. Fluid contains fat, rich in cholesterol, derived from ruptured lymph varix into the tunica. It is often difficult to differentiate from primary hydrocele. In chylocele, chylous fluid collects in tunica vaginalis which may show microfilaria (**Fig. 18.18**).

Complications of Hydrocele

Infection; pyocele (suppurated hydrocele); haematocoele; atrophy of testis; infertility; rupture; hernia of the hydrocele sac through dartos (**Fig. 18.19**).

Differential Diagnosis

Inguinal hernia; epididymal cyst; spermatocele; testicular tumour; scrotal oedema.

Conditions which cause loss of testicular sensation

Testicular tumour
Lepra orchitis
Syphilitic orchitis
Chronic haematocoele



Fig. 18.19: Hernia of hydrocele can occur through dartos muscle.

Haematocoele

It is collection of blood/clot in the tunica vaginalis testis. It may be due to trauma or bleeding in an existing hydrocele. Blood gets clotted, organised and later may be calcified. Eventually it causes testicular atrophy.

Types: Recent haematocoele—It is due to rupture of one of the vessels in the tunica causing bleeding into the sac. It often may be due to aspiration of a hydrocele. It may be precipitated by trauma also.

Clinical features: Sudden onset of pain, swelling after a history of trauma; tender, warm, fluctuant, but non-transilluminant; occasionally aggressive testicular tumour mimics presentation of acute recent haematocoele. US of scrotum is done in such suspected cases to rule out neoplasm and also to find out the viability of testis.

Complications: Chronic (old clotted) haematocoele; infection; pyocele.

Chronic or old clotted haematocoele—It is usually due to slow, spontaneous haemorrhage into the tunica vaginalis without any proper history of trauma. It is painless, hard, nontender, nonfluctuant, often calcified swelling, with *loss of testicular sensation*. Because of the constant pressure testicular function and so testicular sensation is lost. It mimics testicular tumour in many aspect (**Fig. 18.20**).

Pyocele

It is collection of pus in the layers of tunica vaginalis. It is often suppurative hydrocele. It can occur in a previously normal tunica or in a preexisting haematocoele or hydrocele which gets infected.



Fig. 18.20: Haematocoele.

Features: Fever, toxicity, tender swelling in the scrotum, with scrotal wall oedema; often in young individuals, it may be difficult to differentiate this from the torsion testis; pus under tension eventually causes infective thrombosis of testicular vessels, leading to nonviability of the testis or testicular gangrene (**Figs 18.21A and B**).



Figs 18.21A and B: Pyocele is suppurative hydrocele.

Cyst of the Epididymis

It is due to the cystic degeneration of: (1) Paradidymis (*organ of Germalde*)—is the commonest cause; (2) Appendix of the epididymis (hydatid of Morgagni); (3) Appendix of the testis; (4) The vas aberrans of Haller. Even though it is of congenital origin, it occurs in middle age. It is tensely cystic, contains clear fluid; often bilateral; they are aggregation of number of small cysts and so multiloculated. They feel like '**bunch of tiny grapes**' situated *behind the body* of the testis. Because of numerous septae they are finely tessellated and brilliantly transilluminant, giving a '**Chinese lantern pattern**'.

Cyst of an appendage of the testis is a unilateral globular rare cystic swelling in the superior pole of the testis which may develop torsion.

Spermatocele

It is a *unilocular acquired retention cyst* derived from blockage of some portion of the sperm conducting mechanism of the epididymis. It is situated in the head of the epididymis, above and behind the body of the testis. Swelling contains *barley water like fluid* which contains spermatozoa. It is soft, cystic and transilluminant. It is often considered by the patient like having additional testis. Aspiration cytology confirms the diagnosis. It is often called as '*third testis*'.

Epididymal cyst	Spermatocele
Congenital	Acquired retention cyst
Behind and above the testis	Behind the body of the testis
Multilocular	Unilocular
Bunch of grapes appearance	Looks like 3rd testis
Clear fluid as content	Barley water fluid contains sperms
Brilliantly transilluminant	Transilluminant
Excision should be avoided in young	Can be excised

Varicocele

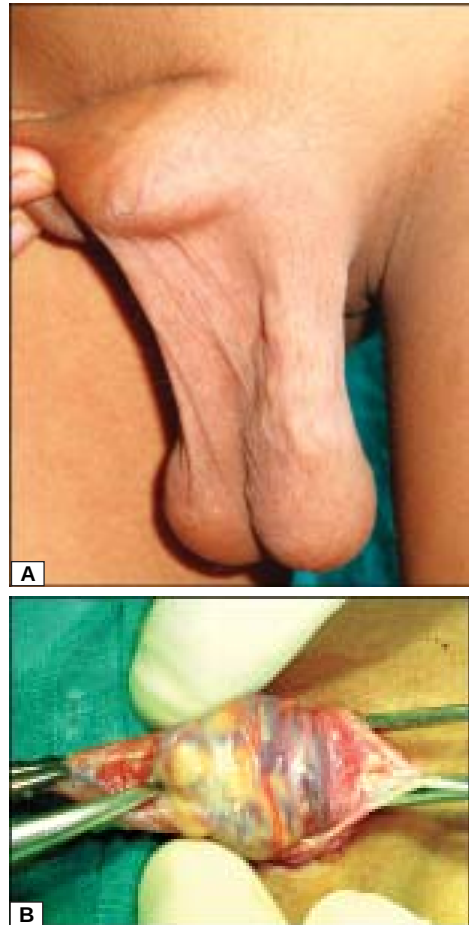
It is dilatation and tortuosity of the pampiniform plexus of veins and so also the testicular veins. Normally there will be plenty of plexus of veins (pampiniform) in the scrotum, which all join together to form about 4-8 veins in the inguinal canal. Above, in the abdominal cavity, in the posterior abdominal wall all join to form

a single testicular vein. On left side, it drains into left renal vein; on the right side it drains into inferior vena cava. Varicocele is common in *tall, thin young men*. More common on *the left side*, but often can be bilateral. Commonly it is idiopathic, may be due to absence or incompetent valve at the junction of left testicular vein and left renal vein causing inefficient drainage of blood. Other reason is, due to perpendicular (right angle) entry of the left testicular vein into the left renal vein. In left sided renal cell carcinoma, tumour proliferates into the left renal vein and blocks the entry of left testicular vein causing varicocele on left side which are *irreducible*. Varicocele causes increased temperature in the scrotum which depresses the spermatogenesis and so *causes infertility* (correctable infertility).

Types of varicocele: *Primary/idiopathic*– 95%: No cause is found. There is incompetence of valves of the testicular vein. It is common on left side as left testicular vein joins left renal vein perpendicularly; left side vein is longer and is liable to get compressed by loaded sigmoid colon. Left renal vein is often compressed between aorta and SMA. *Secondary* – due to specific cause like left sided renal cell carcinoma with a tumour thrombus in left renal vein causing obstruction to venous flow of left testicular vein (**Fig. 18.22**).

Clinical features: Swelling in the root of the scrotum; dragging pain in the groin and scrotum; ‘*Bag of worms*’ feeling; impulse on coughing (thrill feel); on lying down it gets reduced slowly and spontaneously (except in renal cell carcinoma). *Bow sign:* After holding the varicocele between thumb and fingers, patient is asked to bow. Varicocele gets reduced in size. Bowing reduces the blood flow of testicular vein and pampiniform plexus causing reduction in size.

Grading of varicocele: I–small; II–moderate; III–large; IV – severely tortuous. *Subfertility/infertility* are observed in even unilateral varicocele. It is a debate whether it really causes subfertility. Possible causes are – Altered heat exchange mechanism of the scrotum due to varicocele → hyperthermia → inhibition of spermatogenesis. Increased blood flow → increased temperature in the testes → increases the metabolic activity using glycogen storage → depletion of glycogen → injury of parenchyma of testes →



Figs 18.22A and B: Left-sided varicocele and also on table look.

oligospermia, hypoxia of testes. Same cause leads into Leydig cell dysfunction; decreased testosterone levels. Final effect is maturation arrest → poor spermatogenesis.

Investigations: Venous Doppler of the scrotum and groin; US abdomen to look for kidney; Semen analysis.

Funiculitis

It is inflammation of the vas deferens. It is commonly due to filariasis. It can be of gonococcal; tuberculous aetiology. Filarial funiculitis presents as mild pain in the inguinal canal and cord with fever; red, oedematous, shiny skin. Often oedema is so severe that it should be differentiated from strangulated inguinal hernia. Palpation above the deep inguinal ring makes one to

feel the hernial contents whereas in filariasis it is normal. Tuberculous funiculitis is associated with tuberculous epididymitis; having thickened, craggy, beaded feel.

Filarial Epididymo-orchitis

Filarial inflammation begins in globus major making it oedematous, firm, thickened and tender. Eventually testis is involved and becomes tender. Secondary hydrocele or chylocele develops.

Lymph Varix (Lymphangiectasis)

Here lymphatic vessels of the cord get dilated and tortuous due to obstruction by filarial worm. Previous periodic attacks of fever, pain, discomfort are obvious. Presents as soft, cystic, boggy swelling in the inguinal/inguinoscrotal region which has got thrill-like impulse on coughing and gets reduced slowly and spontaneously on lying down. Groin lymph nodes may get enlarged. Dancing filarial worm in US groin is diagnostic. Lymph varix presenting as multiloculated elongated cystic swelling in the cord is called as '*diffuse hydrocele of the cord*'.

Lymph Scrotum and Elephantiasis of Scrotum

It is dilatation and tortuosity of the cutaneous lymphatics of the scrotum. Presents as excess rugosity; vesicles in the scrotal skin which contains clear fluid; often these vesicles may rupture causing lymphorrhagia. Secondary infection occurs; later slowly fibrosis of skin takes place leading into elephantiasis of the scrotum. Initial pitting oedema soon becomes nonpitting, firm, thick skin progressing gradually upwards. It contains lymph logged oedematous tissue with hydrocele inside. Atrophy of testis due to lack of nutrition is common (**Fig. 18.23**).

Orchitis

It is inflammation of the testis. It is commonly associated with inflammation of the epididymis. Hence it is called as *epididymo-orchitis*. Orchitis is due to infection through blood, lymphatics or epididymis. *Causes*: Viral infection—mumps; filarial disease; leprosy; bacterial; brucellosis; infectious mononucleosis. It can be precipitated by retrograde spread due



Fig. 18.23: Scrotal elephantiasis—filarial cause.

to stricture urethra, after prostate or bladder surgery, after instrumentation. Syphilis involves testis—causing formation of gummatous ulcer on the front of the scrotum. *Features*: Pain in the testis often radiates to groin due to associated funiculitis; Fever, tenderness in the testis; Secondary hydrocele is common; Often urinary infection is noticed.

Differential diagnosis: Torsion testis; Testicular tumour. **Syphilitic orchitis**: Syphilis involves *only testis*; never vas deferens. It can be—*bilateral interstitial orchitis seen in congenital syphilis* (causing *pigeon-egg* testes in infants; if infant becomes syphilitic boy then he becomes *lame* (Clutton's joints), *deaf* (neurolabyrinthitis), *blind* (interstitial keratitis), *impotent* (atrophy of testes); *interstitial fibrosis* is bilateral causing gradual destruction of the seminiferous tubules with loss of testicular sensation without any enlarged testis. Testis is dense, rounded hard and mobile—'*billiard testis*'; **Gumma of testis** is commonest type with unilateral painless slowly enlarged hard testis with loss of testicular sensation. Testis is adherent to anterior part of the scrotal skin leading into softening and gummatous ulcer formation. Shotty groin, epitrochlear and popliteal lymph nodes may be palpable.

Epididymitis

Inflammation of epididymis is commonly associated with orchitis—*epididymo-orchitis*. **Causes:** Non-specific, viral like mumps; bacterial; filarial; tuberculosis (It involves mainly epididymis not testis and so ulcer/sinus occurs over the posterior aspect of the scrotum not in front); gonococcal; schistosomiasis. It can be acute or subacute or chronic. Acute when it occurs from retrograde spread involves globus minor first later entire epididymis and testis. Severe pain, oedema scrotum, thickened tender epididymis, secondary hydrocele are common (**Fig. 18.24**). There may be associated prostatitis, urethritis, and cystitis also. Blood born infection involves globus major first. Retrograde spread can occur after prostatectomy, catheterisation, and cystitis.



Fig. 18.24: Epididymo-orchitis with hydrocele on table.

Tuberculous epididymitis: It is commonly due to retrograde spread from tuberculous cystitis. It involves globus minor (tail) first and later entire epididymis and testis in very late cases. Blood spread from lungs directly involves globus major first. Thickened, craggy,

firm nodular epididymis is common. Cold abscess, sinus or undermined ulcer may be present on the posterior aspect of the scrotum. Lesion will be on the anterior aspect in anteverted testis. Scrotal skin loses its normal rugosity with wasting of the tissue under the skin. There is restricted mobility (upward and downward) of testis. Thickened beaded vas (due to tubercles) is typical. Secondary hydrocele develops in 30% cases. 60% will be having renal tuberculosis. Digital examination of rectum (P/R) shows tender thickened palpable seminal vesicles and irregular prostate. Pulmonary tuberculosis is evident in 50% of cases.

Diffuse Lipoma of the Cord

It is a rare soft lobulated diffuse lipoma of the cord involving inguinal portion. It does not show any impulse on coughing.

Undescended Testis

It results from arrest of descent of the testis in some parts of its path to the scrotum. Bilateral undescended testis is called *cryptorchidism* (means hidden testis). *Anorchism*: There is complete agenesis of testis. These two can be differentiated by HCG test.

Embryology

Normally kidney ascends, testis descends during development. Primitive testis develops from the genital fold which is attached to the posterior abdominal wall by mesorchium. It lies below the developing kidneys. Wolffian duct develops into epididymis and vas deferens. A fold of peritoneum develops at the junction of vas deferens and epididymis which can be traced down upto the developing phallus (scrotum) and is called as *gubernaculum*. Along with some hormonal factors, the muscular fibres in the gubernaculum assist in the descent of testis. During 9th month of gestation testis reaches deep inguinal ring. Later just before or after delivery it descends into the scrotum.

Incidence

In premature infants - 30%. In full term infants - 4%. In later childhood - 2%. Right testis is involved more commonly in 50% cases, left alone in 30% cases,

bilateral in 20% cases. It is due to gubernacular dysfunction, lack of HCG, *Prune Belly* syndrome, familial.

Pathology: Up to the age of six, microscopic changes are uncommon. After that, testis gradually atrophies, reduces its external as well as internal secretory activity. Eventually grossly immature epithelial elements with irreversible destructive changes of the germinal epithelium occur.

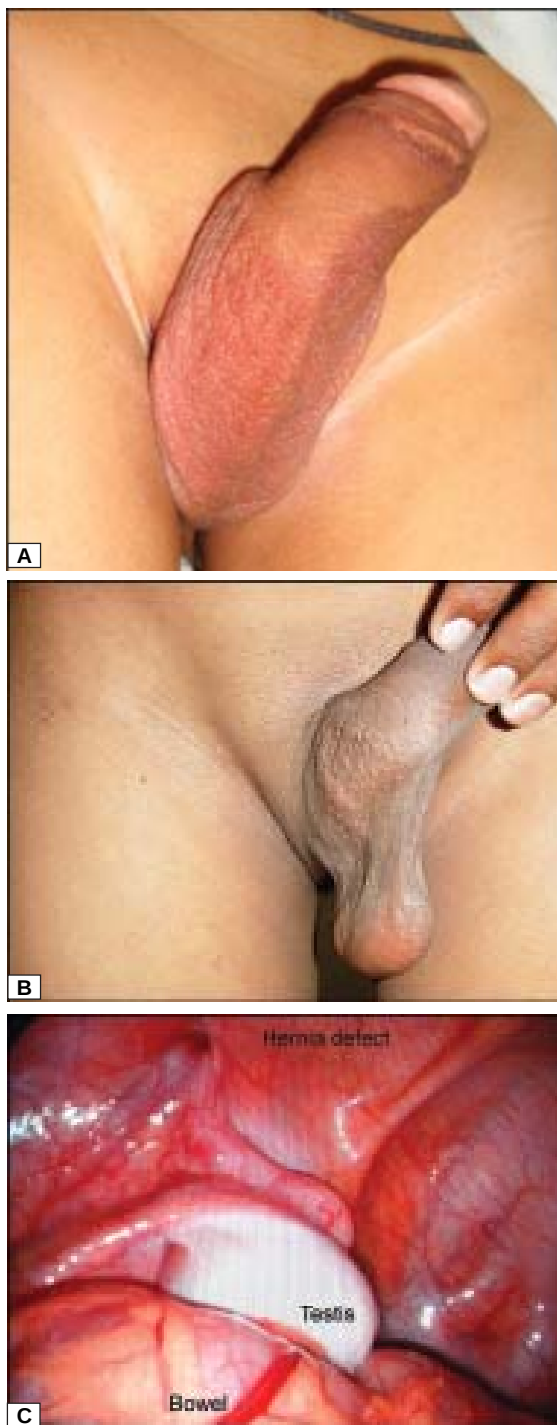
Different location of testis: In the abdomen just above the internal ring, extraperitoneally; in the inguinal canal; in the superficial inguinal pouch. Bilateral undescended testes which are clinically impalpable is called as **cryptorchidism**. Scrotum is *not fully developed* and testis cannot be brought down manually to the bottom of the scrotum in undescended testis.

Differential Diagnosis

Retractile testis.

Complications of undescended testis: Sterility; trauma and pain; an associated indirect inguinal hernia (70%); torsion testis; epididymo-orchitis (as the pain will be high up, it mimics acute appendicitis); testicular atrophy; *Malignant transformation in undescended testis is 20 times more common* than in normally descended testis. It is higher in abdominal than in inguinally located testis. *Seminoma* is the commonest malignancy in undescended testis. The testis which has normally descended on other side (in case of unilateral undescended testis) is also more prone for malignant transformation than normal individual. US abdomen and groin; gonadal venogram and laparoscopy are diagnostic methods available (**Figs 18.25A to C**).

Note: Undescended testis is a terminology which is commonly used in general but by proper definition correct terminology is different. But students should use undescended testis as the required terminology. Any problem in the mode of descent of testis is called as *imperfectly descended testis*. *Maldescended testis* is the testis that cannot be made to touch the bottom of the scrotum. A *true undescended testis* is the one which has not moved from its origin just below the kidney and such situation is extremely rare. *Cryptorchidism* in a male is a situation where both



Figs 18.25A to C: (A) Undescended testis—bilateral; (B) Unilateral; (C) Laparoscopic view of undescended testis.

testes are hidden and impalpable, probably above the level of deep inguinal ring in the retroperitoneum. Maldescended testis includes either undescended testis or ectopic testis.

Ectopic Testis

Lockwood Theory

Eventhough there are multiple gubernaculum, scrotal tail normally gets activated better and stronger and so testis is brought down to the scrotum. In *ectopic testis* scrotal tail weakens or ruptures and so one of the other accessory tails will act stronger and pulls the testis according to their site.

Different Sites

(1) Superficial inguinal pouch (commonest site); (2) Perineum; (3) Root of the penis; (4) Femoral triangle (Thigh). Here testis is functioning normally and of normal size. It is more prone for trauma and can cause psychological problem. Scrotum is not properly developed.



Fig. 18.26: Orr chair test.

Retractile Testis

It is due to the strong overaction of cremaster, as a result testis is pulled up, to stay near the external ring and often mistaken for undescended testis. Here testis is normally developed, can be pulled down to the bottom of the scrotum properly. Scrotum is also fully developed. Child is made to sit on a chair with feet kept on the chair; knees fully flexed and brought over to chest wall; causing pressure on the inguinal canal downwards pushing retractile testis down into the scrotum – *Orr chair test* (Fig. 18.26).

Torsion of the Testis

It is an emergency condition of the testis, wherein the testis twists (rotates) in its axis compromising its blood supply. If not intervened and rectified within 12-24 hours, testis will become gangrenous. Right testis rotates in clockwise direction whereas left rotates in anticlockwise (Fig. 18.27).

Predisposing factors: (1) Inversion of the testis. (2) High investment of the tunica vaginalis which acts like a mesentery through which testis rotates. Here testis hangs like a *clapper in bell*. (3) Presence of

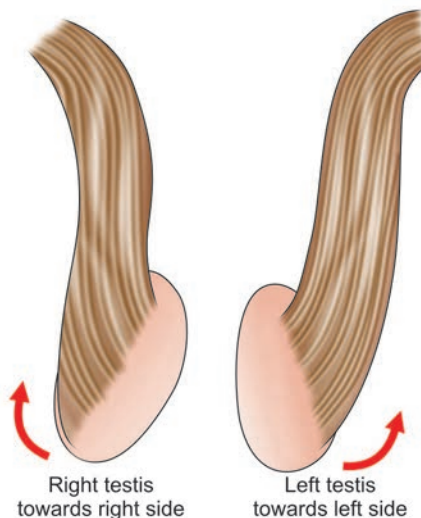


Fig. 18.27: Right testis torsion occurs towards right side (clockwise); left towards anticlockwise (left)

gap between the body of the testis and epididymis as a result of which testis twists over epididymis. (4) Heavy straining often precipitates torsion due to vigorous contraction of the cremaster which is attached spirally.

Clinical features: It occurs in children and adolescents. It presents with sudden onset of pain in the scrotum, groin and lower abdomen. Vomiting due to pylorospasm is common. Tenderness, redness, and oedema of the scrotal skin (**Figs 18.28A to C**). Torsion occurring in an imperfectly descended testis is impossible to differentiate it from strangulated hernia. Absence of testis in the scrotum may give a clue.

Deming's sign: Affected testis is positioned high because of twisting of cord and spasm of cremaster muscle.

Angell's sign: Opposite testis lies horizontally because of the mesorchium between testis and epididymis and is usually bilateral.

Differential diagnosis: (1) Acute epididymo-orchitis-elevation of the scrotum relieves the pain of acute epididymo-orchitis but aggravates in case of torsion testis (*Prehn's sign*). (2) Strangulated inguinal hernia. (3) Other structure in scrotum which can undergo torsion is '*Appendage of testis*'. If the patient is able to walk to clinician with feature of torsion then this condition has to be thought of whereas in torsion testis the pain is so severe that the clinician is summoned to the patient's bed. Secondary hydrocele of the torsion testis is serosanguinous.

Testicular Tumours

It accounts for 1% of all malignant tumours; 99% of testicular tumours are malignant.

Predisposing factors: Undescended testis, Klinefelter's syndrome and testicular atrophy.

Classification: (1) Seminoma - 40%. (2) Teratoma-32%. (3) Seminoma + teratoma - 14%. (4) Interstitial tumours-1.5% (Leydig cell tumour (musculinises; Sertoli cell tumour feminises). (5) Lymphomas - 7%. (6) Others.

Histological classification: (1) *Germ cell tumour* – Seminomatous: classic/spermatocytic/anaplastic. Non-seminomatous: embryonal carcinoma/teratoma/choriocarcinoma/yolk sac tumour. (2) *Sex cord tumours*: Leydig cell tumour; Sertoli cell tumour. (3) *Combined germ cell and gonadal stromal tumour*. (4) *Adnexal and paratesticular tumour*. (5) *Others* – Carcinoids, lymphomas, secondaries.



Figs 18.28A to C: Typical torsion testis which is elevated with redness and adherent skin.

Seminoma testis: It starts in the mediastinum of the testis. Grossly it is lobulated, fleshy, homogenous, creamy or pinkish in colour and it compresses adjacent testicular tissues. Histologically, malignant cells resemble spermatocytes which are *clear cells*, with *lymphocytic* infiltration. It spreads through testicular lymphatics into the para-aortic lymph nodes and then to left supraclavicular lymph node. Through blood, it spreads to lungs, bone, brain, liver. Seminoma is further classified as typical (classic) which is commonest; spermatocytic (in old age); anaplastic; atypical.

Teratoma: It arises from totipotent cells, i.e. ecto, meso, endoderms. Grossly tumour surface is irregular, cut section shows solid and cystic spaces with areas of haemorrhage. It often contains gelatinous fluid and cartilaginous nodules (**Fig. 18.29**). Histologically there are four types: (1) *Teratoma differentiated* – (1%); (2) *Teratoma intermediate* - 30% common - Two subtypes are A and B (more malignant); (3) *Teratoma anaplastic* – 15% - secretes alpha feto protein (AFP); (4) *Teratoma trophoblastic* - 1% - It shows high levels of β HCG (normal level is 100 IU).



Fig. 18.29: Testicular teratoma fungating through the skin.

Interstitial Cell Tumour

Leydig cell tumour (2%) masculinises; Prepubertal tumour shows excessive output of androgens causing sexual precocity, extreme muscular development and may mimic infant hercules.

Sertoli cell tumour (1%) feminises; Post-pubertal tumour commonly arising from sertoli cells causes feminising effect with gynaecomastia, loss of libido and aspermia.

Clinical features: Enlargement of testis; fullness and heaviness in the scrotum; pain in the testis (30%); testis will be enlarged, firm, heavy, with loss of testicular sensation; secondary hydrocele is common. Cremaster is hypertrophied and thickened. Vas, prostate and seminal vesicles are normal. It can spread to cord tissues making it nodular and hard. Often in epigastric region para-aortic lymph nodes may be palpable as hard, nodular, nontender, nonmobile, vertically placed, resonant mass (not moving with respiration). There may be haemoptysis, altered breath sounds and pleural effusion due to lung secondaries; Bone pain and tenderness due to secondaries in bone; Nodular secondaries in the liver. Occasionally it may mimic acute epididymo-orchitis or acute haematocele. Gynaecomastia may be present in few teratomas.

Hurricane type is very aggressive, highly malignant testicular tumour which is more often fatal in few weeks. Rarely, if tumour comes out of the tunica albuginea (tunica albuginea is resistant for malignant cell infiltration), then scrotum gets infiltrated and spread can occur to inguinal lymph nodes.

Differential diagnosis: Acute and chronic haematocele; acute epididymo-orchitis; syphilitic orchitis; Lepa orchitis.

Sign of vas: To differentiate tumour from infection - in testicular tumours vas is normal, cord structures may become bulky because of cremasteric hypertrophy whereas in infection vas is thickened, beaded, and tender.

Investigations: **No FNAC; No scrotal approach; No incision biopsy.** Through inguinal approach, cord and testis are exposed. A soft clamp is applied to the cord at or above the level of the deep ring so as to prevent dissemination through blood. Frozen section biopsy is done from the suspected area. If tumour is positive *high orchidectomy is done (Chevassou manoeuvre)*. Tumour markers β HCG, AFP are increased in teratoma; Chest X-ray, CT chest; US abdomen; US scrotum to see echogenicity of testis and tumour within.

Staging of testicular tumours:

Lesion confined to testis
 Nodes involved below the diaphragm
 Nodes involved above the diaphragm
 Distant (blood) spread

TNM staging

T ₀	No evidence of tumour
T _{is}	Carcinoma in situ
T ₁	Tumour limited to testis and epididymis. Vascular/lymphatic invasion not present. Tumour may invade tunica albuginea but not tunica vaginalis
T ₂	Tumour limited to testis and epididymis with vascular/lymphatic invasion. Or tumour extends through the tunica albuginea with involvement of tunica vaginalis
T ₃	Tumour invades to spermatic cord with or without vascular / lymphatic invasion
T ₄	Tumour invades to scrotum with or without vascular/lymphatic invasion
N ₀	Regional nodes not involved
N ₁	Single / multiple nodes – not more than 2 cm in size
N ₂	Regional nodes – between 2-5 cm
N ₃	Regional nodes > 5 cm

Extravasation of the Urine

It may be superficial or deep.

Superficial

It is either due to bulbar urethral injury or due to bursting of periurethral abscess after urethral stricture. Once urine extravasates due to disruption of full thickness of the urethra anteriorly, it collects in superficial perineal space. This space is a closed cavity all around except anteriorly where it communicates with scrotal subcutaneous tissue deep to fascia Colles, penis between superficial fascia and deeper Buck's fascia, in the anterior abdominal wall deep to Scarpa's fascia. It does not spread to thigh and ischiorectal space as



Fig. 18.30: Superficial extravasation of urine.

Scarpa's fascia is attached firmly to fascia lata of thigh. Superficial perineal space is closed above by inferior fascia of perineal membrane; below by fascia of Colles; laterally by ischiopubic rami. It is open and communicating only anteriorly. Entire scrotum, penis and often lower abdominal wall are swollen containing urine. It is painful; patient cannot pass urine through urethra; Has severe pain and shock due to pelvic injury. Often sepsis occurs and skin sloughs of leading into urinary fistulas.

Deep

Urine spreads upwards into the extraperitoneal space of the pelvis around the bladder and prostate into the anterior abdominal wall causing deep extravasation of the urine. Here rupture of urethra is at membranous part of the urethra much more proximal than superficial type (**Fig. 18.30**).

Examination of Male External Genitalia

History

Phimosis, hypospadias are seen in infants and children. Carcinoma of penis is seen in adult and old age.

Muslims and Jews undergo early circumcision and so they are immune from developing carcinoma penis.

Inability to retract foreskin in a child as history given by mother or ballooning of the prepuce during urination or visible pinhole meatus are common. Pinhole meatus may be congenital commonly or acquired due to balanoposthitis or meatal ulcer.

History of paraphimosis – Inability to place back the retracted prepuce. It may be precipitated in a patient with mild phimosis by act of intercourse. Paraphimosis may be painful.

History of ulcer in the penis should be asked in detail. Its location, duration, progress, pain, discharge, bleeding, urinary symptoms, change in the stream of urine are important. *Chancroid* is due to *Haemophilus ducreyi* (soft sore) develops in 4 days after exposure as a painful, tender ulcer. *Syphilitic Hunterian hard chancre* appears 4 weeks after exposure. Small painless ulcer often disappears unnoticed in *lymphogranuloma venereum* (LGV). Painless vesicle or papule later forms a granulomatous ulcer in *granuloma inguinale* (Donovan ulcer, lymph nodes are involved). Progressive painless ulcer may be carcinomatous ulcer.

History of discharge, its duration, site of discharge, foul smelling or not should be asked.

History of sexual contacts is very important in all these ulcers.

History of pain may be in the glans, in the ulcer, in the urethra, during micturition (urethritis, stone, prostatitis) or may be independent of act of micturition (herpes, carcinoma, balanoposthitis, etc).

History of fever may be due to infection.

History of swelling in the groin should be noted. Carcinoma of penis can spread into the inguinal lymph nodes causing secondaries. Lymph nodes also can be involved in syphilis, lymphogranuloma inguinale. Often there will be pain, suppuration, ulceration or fungation in the groin which should be asked in detail in history.

General Examination

Anaemia, clubbing, jaundice, nutrition should be assessed. Pulse, blood pressure should be recorded.

Local Examination

Inspection

Inspection of prepuce:

Phimosis, paraphimosis should be looked for by holding the penis properly using a gloved hand. Prepuceal swelling or oedema should be observed. Pinhole meatus/ulcer over the prepuce should be observed. If ulcer is present, its size, shape, edge, floor, discharge, number should be noted. Raised everted edge is a feature of carcinoma. Features of different *premalignant conditions* like—leukoplakia, Paget's disease of glans/inside the prepuce, *Erythroplasia of Querat* which is a red flat area in glans or inner aspect of the prepuce should be looked for. Multiple warty like projections may be condyloma acuminata. Venereal warts are moist with foul smelling discharge. Posthitis (inflammation of prepuce) or balanoposthitis (inflammation of prepuce along with glans) with discharge is obvious on inspection. Ballooning of prepuce while micturition is obvious on inspection. Altered urinary stream occurs in carcinoma penis which



Fig. 19.1: Pinhole meatus causing phimosis. Ballooning of prepuce is common.



Fig. 19.2: Paraphimosis after passing urinary catheter. After passing urinary catheter, prepuce should be placed backwards otherwise paraphimosis will develop.

is close to the meatus or rarely involving the urethra (**Figs 19.1 to 19.3**).

Urethra should be examined for congenital anomaly. If urethral meatus opens more proximally along the ventral aspect, it is called as *hypospadias*. If it opens proximally over the dorsal aspect it is called as *epispadias*. Based on position it is categorised as *glandular* (glans); *coronal*; *penile*; *perineal* with bifid scrotum. Urethral meatus may not be visible in carcinoma of the glans which is close to the meatus.

The body of penis is inspected for ulcer, swelling, etc. Urethral papilloma from fossa navicularis may protrude from external urethral meatus causing



Fig. 19.3: Erythroplasia of Querat. It is a premalignant lesion.



Fig. 19.4: Hypospadias.

haematuria and pain. Often urethral stone exuding just at the external meatus may be observed (**Figs 19.4 and 19.5**).

The groin is inspected for visible swelling as enlarged lymph nodes (**Fig. 19.6**).

Palpation

Palpation should be done by wearing gloves.

One should look for tenderness, and warmth, palpate the ulcer edge and base is palpated for induration, extent of induration, whether bleeds on touch. Prepuce may not be retracted back when there is carcinoma under prepuce. Careful feeling of the prepuce and glans together will appreciate the indurated swelling under the prepuce. Such patient might require circumcision or dorsal slit to visualise the



Fig. 19.5: Stone in the meatus which is visible. Stone was extracted later.



Fig. 19.7: Carcinoma of penis – proliferative type.



Fig. 19.8: Carcinoma of penis – ulcerative induration of glans near corona in front.



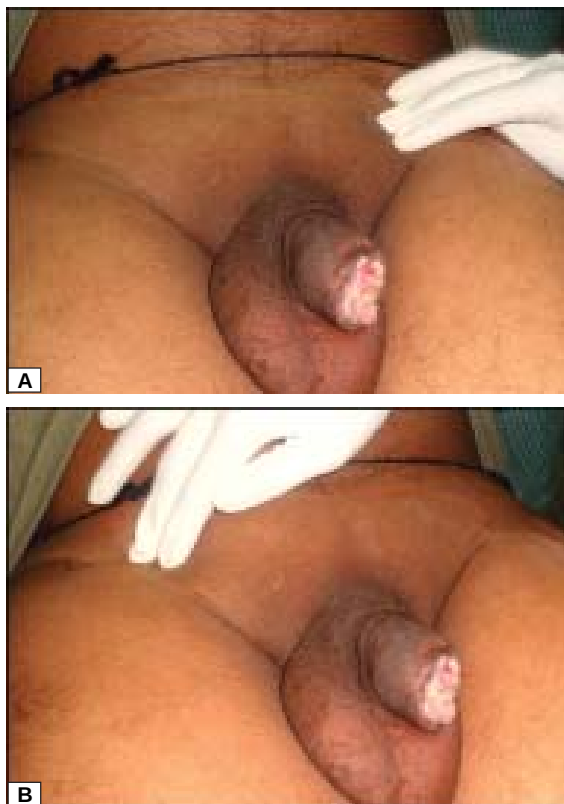
Fig. 19.6: Carcinoma of penis earlier operated—total amputation of penis was done with perineal urethrostomy. Left side inguinal lymph nodes are enlarged with fungating secondaries.

lesion. Urethral discharge can be collected by milking the penis and discharge should be sent for culture, and cytology.

Entire body of penis should be palpated for extent of induration. Urethra should be palpated (**Figs 19.7 and 19.8**).

Palpation of Lymph Nodes

Horizontal group of inguinal lymph nodes or Cloquet's deep node (from glans) may be enlarged. Its size, number, surface, consistency, tenderness, mobility, fixity should be checked. Iliac nodes above the inguinal ligament may be involved due to spread from inguinal nodes. Involvement of urethra also can cause enlargement of iliac node. In 50% cases initially the enlargement may be due to infection only. Urethral involvement is probably due to infection or tumour (**Figs 19.9A and B**).



Figs 19.9A and B: Carcinoma of penis – palpation of inguinal lymph nodes both sides.

Disorders of Penis

Phimosis (Greek- a stooping up; a closure)

It is inability to retract the prepuce over the glans. End of the prepuce is very narrow, often like pinhole (*pinhole meatus*) (**Fig. 19.10**).

Causes: 1. Congenital - here the child has pinhole meatus and ballooning of prepuce occurs when child urinates. 2. Balanitis (inflammation of glans) and balanoposthitis (inflammation of glans, prepuce and sac). It is common in diabetics.

Problems due to phimosis: Recurrent balanoposthitis; paraphimosis; ballooning of prepuce skin; retention of urine; formation of prepuceal calculi due to smegma collection in prepuceal sac; carcinoma of penis later.

Paraphimosis

It is inability to place back the retracted prepuceal skin over the glans. It causes ring like constriction



Fig. 19.10: Phimosis.

proximal to the corona and prepuceal skin. As a result the glans will be swollen, oedematous with severe pain and tenderness. Retracted narrow prepuce at corona, acts as a tight constricting ring which blocks the venous blood flow causing congestion and, oedema of the glans (**Fig. 19.11**). It is very painful and tender. Often glans undergoes necrosis or becomes gangrenous. Paraphimosis is often precipitated by sexual intercourse or iatrogenically after urethral catheterisation.



Fig. 19.11: Paraphimosis. Note the constriction band and oedema.

Balanoposthitis (Greek)

It is the inflammation of glans and prepuce. Inflammation of prepuce is called as *posthitis*; inflammation of glans is called as *balanitis*. It is seen in diabetes mellitus, candidiasis, venereal diseases like syphilis, herpes genitalis or drug induced. It can cause phimosis (Fig. 19.12). In adult there may be underlying carcinoma of penis. Pain, swelling, discharge and discomfort are the features. Itching, creamy intolerable smell, difficulty to retract prepuce, multiple fissuring in the tip, itchy vesicles with shallow painful erosions of herpes are other features.



Fig. 19.12: Balanoposthitis causing phimosis.

Chordee

It is fixed bending of glans penis, more obvious during erection. It can be ventral or dorsal. Ventral chordee is associated with hypospadias. During circumcision, if ventral skin is excised more than needed, it will cause chordee. Dorsal chordee is rare and is associated with epispadias (Fig. 19.13).

Priapism

It is persistent, painful erection of penis. Corpora cavernosa are filled with blood due to defective venous



Fig. 19.13: Chordee with hypospadias.

drainage. Glans and corpus spongiosum are not involved.

Causes: Idiopathic thrombosis of corpora cavernosa; Thrombosis of prostatic venous plexus; Sick cell disease; Leukaemia; Secondary deposits in corpora cavernosa; Spinal injury or diseases and organic diseases of central nervous system.

Peyronie's Disease (Induratio-penis plastica)

It is development of fibrous tissue plaque on the covering of corpus cavernosum and later involving its full extent resulting in induration of corpus. It is a slowly progressive disease of uncertain aetiology, may be due to old trauma, often associated with *Dupuytren's contracture*, *retroperitoneal fibrosis* and *plantar fasciitis*. Initial active phase has painful erection with changing deformity of penis, followed by quiescent phase where there is disappearance of painful erection with development of deformity which is painless. Later indurated plaque is noticed with penile shortening and erectile dysfunction.

Rams Horn Penis

It is due to filarial involvement of penis where it becomes thick and distorted resembling horn of a ram. It is actually elephantiasis of penis (Fig. 19.14).



Fig. 19.14: Penile oedema and scrotal oedema.

Hypospadias

It is the commonest congenital malformation of urethra wherein external meatus is situated *proximal* than normal, over the *ventral* (under) aspect of the penis.

Classification: (1) Glandular: Meatal opening in glans. It is the commonest. (2) Coronal. (3) Penile. (4) Penoscrotal. (5) Perineal with split scrotum and meatus is 3 cm in front of the scrotum. This is associated with bilateral undescended testes.

Features: (1) Absence of urethra and corpus spongiosum distal to abnormal urethral orifice. (2) Bowing

or bending of penis distal to abnormal urethral opening (chordee) with poorly developed prepuce over inferior aspect. (3) Urine soakage over the scrotum with dermatitis and infection. (4) Associated congenital anomalies are known to exist. *In hypospadias circumcision is contraindicated as prepuce skin is required for future urethroplasty.*

Epispadias

Here urethra opens on the dorsum of the penis proximal to the glans. Abdominopenile is the commonest type. Occasionally it can be glandular or penile. It is associated with dorsal chordee, ectopia vesicae, urinary incontinence, and separated pubic bones (**Fig. 19.15**).

Carcinoma Penis

It is commonly *squamous cell carcinoma*, but melanoma, adenocarcinoma from Tyson's gland, basal cell carcinoma and secondaries may also occur.

Aetiology: Chronic balanoposthitis, phimosis; sexually transmitted diseases; leukoplakia of glans; long standing genital warts; *Paget's disease* of penis (*Erythroplasia of Querau* is persistent rawness of glans penis); condyloma acuminata (human papilloma virus); balanitis xerotica obliterans; HIV infection. *Circumcision during infancy confers total immunity against carcinoma penis.* It is common in Asia and Africa.

Pathology: *Infiltrating type* occurs in a preexisting leukoplakia; *Papilliferous type* eventually attains a large size forming fungating foul smelling lesion which



Fig. 19.15: Extrophy of bladder with epispadias.

often gets infected. Glans penis is the commonest site (coronal sulcus for basal cell carcinoma).

Spread: Through lymphatics to the horizontal group of inguinal lymph nodes which become nodular and hard. Lymph nodes on both sides can get involved. Later external iliac group are involved (above and on medial aspect of the inguinal ligament). Once inguinal lymph nodes are fixed it causes severe excruciating pain and lymphoedema. *Fixed lymph node status indicates the advancement of the disease.* It may erode into the femoral vessels causing torrential haemorrhage and death. Carcinoma from penis and glans spread to inguinal lymph nodes and then to external iliac lymph nodes. From glans it also spreads to Cloquet lymph node which is located in femoral canal. Carcinoma from shaft of penis can spread directly to the external iliac lymph nodes. It spreads proximally to the body of penis causing induration. Urethral meatus may get involved causing alteration in urinary stream. *It is a locoregional malignant disease. Blood spread is rare.*

Clinical features: In an adult, recent onset of phimosis should give suspicion of carcinoma penis. Lesion is painless initially but later becomes painful due to secondary infection often accompanied by discharge which is foul smelling, purulent and irritating. Altered urinary stream; everted edge, ulcer, fungation and induration, often extending into the body of penis are other features (**Fig. 19.16**). Palpable hard, nodular inguinal lymph nodes on both sides may be present. External iliac lymph nodes may be palpable. Pain, oedema, tenderness, redness develops once infection occurs. Incidence is less than 1% of male carcinomas; glans – 65%; prepuce – 20%; corona, shaft – 10-15%. Buck's fascia is resistant for initial infiltration; urethral involvement only in late cases.

Investigations: *Edge biopsy* from the lesion shows squamous cell carcinoma with epithelial pearls.

Broder's classification: (1) Grading: Very well differentiated (75% epithelial pearls); Well differentiated (50-75%); Moderately differentiated (25-50%); Undifferentiated (25%). (2) *Only FNAC of lymph node* (No open biopsy for lymph nodes). (3) U/S abdomen to see external iliac lymph nodes. (4) **SLNB-Cabana sentinel node** is located above and medial to the



Fig. 19.16: Carcinoma penis.

junction of saphenous and femoral vein. It is the first node to get involved in carcinoma penis. So this Sentinel Lymph Node Biopsy (SLNB) after isosulphan blue dye injection into the primary is done to decide for the necessity for ilioinguinal block dissection.

Staging of carcinoma of penis

Jackson's staging of carcinoma penis

Stage I—Tumour involving only glans/prepuce / both	90% five year survival
Stage II—Tumour extending into body of penis	70%
Stage III—Tumour having mobile inguinal nodes	50%
Stage IV —Tumour spreading to adjacent structures / fixed nodes	5%

TNM staging

T ₀	No primary tumour
T _{is}	Carcinoma in situ
T ₁	Tumour < 2 cm without deep invasion
T ₂	Tumour between 2-5 cm with minimal deep invasion
T ₃	Tumour > 5 cm with deep invasion / urethral spread
T ₄	Tumour spread to adjacent tissues
N ₀	No nodal spread
N ₁	Mobile regional nodes – unilateral
N ₂	Mobile regional nodes – bilateral
N ₃	Fixed regional nodes
M ₀	No distant spread
M ₁	Distant spread present

Note:

Dressler's quadrangle – upper border is formed by line joining anterior superior iliac spine and pubic tubercle; laterally line joining anterior superior iliac spine and a point 20 cm below it; medially pubic tubercle and a point 15 cm below it. Nodal block dissection for carcinoma penis should cover this area adequately.

Buschke-Lowenstein Tumour

It is verrucous carcinoma of penis (5-15% common). It is a curable malignancy; it is locally destructive; locally invasive. It is large exophytic, dry, verrucae like growth. It neither spreads through lymphatics nor through blood. HPV 6/11 viral aetiology is proposed (Fig. 19.17).



Fig. 19.17: Verrucous carcinoma of penis.

Venereal Warts/Papillomas

It is the commonest benign lesion which can occur in uncircumcised or circumcised individuals. Sites are glans, corona, frenulum, and urethral meatus. It is sexually transmitted disease where trauma occurs during intercourse. Human papilloma virus is the cause.



Fig. 19.18: Genital warts.

These warts are moist, multiple, with serous discharge. Intraepithelial neoplasia and carcinoma of penis may develop in these lesions at later period (Fig. 19.18).

Other Conditions*Morgagni Follicles Infection*

These are pair of follicles which open laterally behind the lips of external urethral meatus. Once it gets infected only, these openings are seen as exuding pus. Often it is seen in urethritis.

Tyson's Gland Infection

Tyson glands are pair of sebaceous glands which secrete smegma which are located on either side of the frenum and ducts open into the prepuce sac. When infected, presents as tender firm swellings on the undersurface of the glans on lateral aspect, usually as a complication of gonococcal urethritis.

Meatal Ulcer

It is seen in young boys usually 1½ years after circumcision. Abrasions over the exposed unprotected meatal mucosa by napkins cause ulceration and scabbing. It causes small red ulcer in the meatus which often heals eventually causing meatal stenosis that often leads into retention of urine. Shortened anteroposterior diameter of meatus causes an acquired pinhole meatus. Secondary urinary infection is common.

Examinations in Chronic Abdominal Conditions

Chronic abdominal conditions comprises of vast number of diseases. Often diagnosing and managing many of them is a clinical challenge to a surgeon. Exact clinical approach and a brief outline of different conditions are discussed here. Detailed discussion is beyond the scope of this book. Students are requested to refer *SRBs Manual of Surgery, 3rd edn* or any other surgical textbooks for explanations.

History taking begins with—

Name:

Age:

Sex:

Occupation:

Address:

Congenital pyloric stenosis occurs in newborn. Duodenal ulcer occurs before the age of 35 years. Gastric ulcer occurs after 35 years. Carcinoma stomach occurs in old age. Chronic pancreatitis, gallstone diseases and hiatus hernia occurs in middle aged.

Congenital pyloric stenosis is common in male infants. Peptic ulcer, carcinoma stomach is common in males. Gallstone disease, hiatus hernia is common in females.

Peptic ulcer is more common in professionals and executives. Old dictum '*Hurry; Worry; Curry*' is probable cause for peptic ulcer in India.

Gallbladder disease is more seen in north east India like Bihar. Peptic ulcer is more common in south India.

History

History of Present Illness

Pain

Pain in chronic abdomen may be sudden, colicky, discomfort like, aching, etc. Pain is the one to which patient comes for consultation with the surgeon.

Duration: Duration of pain often suggests the duration of the disease commonly but not always. Peptic ulcer disease, chronic cholecystitis, chronic pancreatitis may be of long duration. **Periodicity** of pain is important. It is seen in peptic ulcer disease. Patient develops pain for certain period of time like few weeks or months; later for certain period patient is symptom free for few weeks or months. Peptic ulcer pain may be seasonal. Chronic diseases are usually of long duration.

Site: Patient should be asked to point out the site of pain with one finger. It may give clue about the origin of the pain. Often pain is vague and diffuse in nature; it may not be possible to pinpoint the site of pain. Duodenal ulcer pain is pointed in duodenal point 2.5 cm right and above the umbilicus. Gastric ulcer pain is in epigastrium in midline or left sided. Pain of chronic cholecystitis is towards right side lateral to right rectus muscle in right hypochondrium.

Radiation of pain: Penetrated peptic ulcer pain radiates from epigastrium to back. Patient with chronic pancreatitis also develops radiating pain to back. Anastomotic ulcer pain is on the left of umbilicus (as stoma is towards left side) which radiates to left iliac fossa or to back.

Relation with food: In duodenal ulcer, pain is relieved by food intake probably due to neutralisation of acid in the stomach. In gastric ulcer, pain increases after taking food. Pain appears early within half an hour after food intake in gastric ulcer, in 3 hours after food intake in duodenal ulcer. Pain on empty stomach is called as '*hunger pain*'. It is a feature of chronic duodenal ulcer. It usually occurs in early morning. Patient gets up early morning due to pain. Pain of carcinoma stomach is continuous without any relation to food.

Relieving factor: Pain is relieved by taking food in duodenal ulcer. In gastric ulcer pain is relieved after

vomiting or induced vomiting. Patient develops pain after food and so puts his fingers over the pharynx and induces vomiting after which pain is relieved.

Nature of the pain: One should ask whether pain is mild or severe. Whether it is burning or griping or colicky type of pain. Often pain is more by movements. Initial periodicity of pain may change to become continuous type of pain if duodenal ulcer causes pyloric stenosis or gastric ulcer causes *tea-pot or hour glass contracture*.

Nausea and Vomiting

Feeling (sensation) imminent desire of vomiting is called as *nausea*. It may or may not proceed into vomiting. It is observed in chronic diseases like pancreatitis, carcinoma of stomach, peptic ulcer with complications, hepatitis and chronic cholecystitis. It can occur in carcinoma of pancreas, small bowel diseases, subacute obstruction by diseases like abdominal tuberculosis. Vomiting is a feature of pyloric obstruction, gastro-intestinal irritation. *Vomiting* is forceful oral expulsion of gastric contents. *Regurgitation* is appearance of previously swallowed food in the mouth.

Nature and quantity of vomitus: It is important to ask content, colour, quantity, smell of vomitus. Vomitus may contain undigested food particles, blood, coffee ground coloured material. Pyloric stenosis causes projectile vomiting containing undigested food. Bleeding peptic ulcer, oesophageal varices, carcinoma can cause haematemesis. Large quantity, rapid bleed causes frank blood in the vomitus. Small quantity of blood mixed with acid of stomach forms acid haematin presenting as 'coffee ground' vomitus.

Frequency: Repeated persistent vomiting is observed in pyloric stenosis, gastric ulcer. Vomiting is not a feature in duodenal ulcer.

Relation to food and pain: Vomiting after taking food is a feature in gastric ulcer (in 2 hours). Recurrent late vomiting (evening or 6-8 hours after food) is a feature of pyloric stenosis. Vomiting is not related to food intake in cholecystitis and pancreatitis. Vomiting or inducing vomiting relieves the pain in gastric ulcer. Vomiting will not relieve pain in cholecystitis, pancreatitis, and carcinoma of stomach.

Haematemesis

Vomiting blood is called as *haematemesis*. Chronic peptic ulcer is the commonest cause (65%). Other causes are acute ulcers, acute erosive gastritis, oesophageal varices, *Mallory-Weiss syndrome*, carcinoma of stomach, gastric polyps, lymphomas, leiomyomas, portal gastropathy, bleeding disorders, pernicious anaemia, thrombocytopenia. *Gastric antral vascular ectasia* is a rare endoscopically confirmed condition which shows segmented dilated vessel meshes in the antral mucosa (*watermelon/tiger stripe stomach*). It is often associated with achlorhydria and hypergastrinaemia; *Osler-Weber Rendu syndrome*, aorto-duodenal fistula, *Crest syndrome* are other rare causes. *Dieulafoy's disease* is gastric arteriovenous malformation which is covered by apparently normal mucosa which occurs in proximal stomach along the lesser curve. It occurs in proximal stomach near OG junction (within 6 cm) along lesser curve (80% of cases). Bleeding often may be severe and torrential. Profuse rapid bleeding causes haematemesis with frank red blood; slow small bleed causes coffee ground vomitus. Haematemesis should be differentiated from haemoptysis. Haemoptysis is blood in the sputum during coughing. Its content, colour should be asked to differentiate properly. Gastric ulcer more often causes haematemesis. In *pseudohaematemesis*, patient initially swallows the blood coming from upper respiratory tract and then vomits it out (**Figs 20.1 and 20.2**).

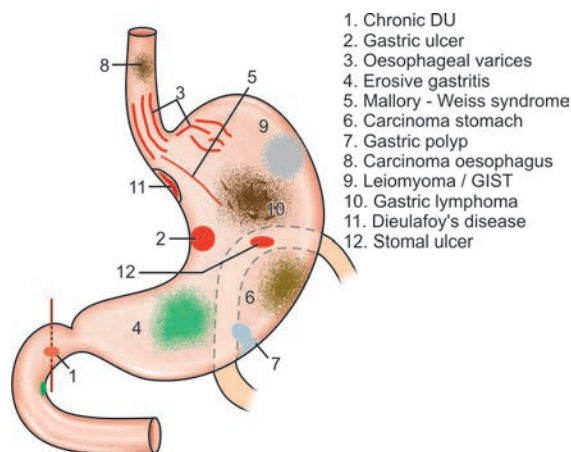


Fig. 20.1: Causes of haematemesis.



Fig. 20.2: Bleeding duodenal ulcer causing haematemesis.

Melaena

It is passing dark, tarry, foul smelling stool per anum. It is a feature of upper gastrointestinal bleed. Common cause is peptic ulcer bleed. Duodenal ulcer more often causes melaena (**Fig. 20.3**).



Fig. 20.3: Typical melaena – black tarry stool.

Flatulent Dyspepsia

Dyspepsia is a vague terminology which includes feeling of fullness in the abdomen after food, belching,

heart burn (*pyrosis*). It may be a feature of gallbladder disease, hiatus hernia, and pancreatitis. *Heart burn* is sensation of warmth or burning situated substernally or high epigastrium radiating to neck or arms. *Belching* is repetitive eructations.

NON-ULCER DYSPEPSIA

- Symptom complex with pain and discomfort in the upper abdomen.
- It occurs in 25% of population – large number
- Anatomical or biochemical abnormalities are not discovered in this condition
- *H. pylori* is not associated with this condition
- Often it lasts for long time decreasing the quality of life
- Differential diagnosis – GERD / acid peptic diseases/ gallstones / pancreatitis / carcinoma
- *H. pylori* eradication is not required and there is no surgical role

Jaundice

Yellowish discolouration of sclera and mucous membrane is called as jaundice. It may be due to neoplasia like carcinoma head of pancreas, periampullary carcinoma, Klatskin tumour, cholangiocarcinoma, nodes compressing porta hepatic, carcinoma of gallbladder, hepatocellular carcinoma, secondaries in liver; biliary stone disease; hepatitis, cirrhosis, pancreatitis, pseudocyst or due to haemolytic causes. In broad day light jaundice is confirmed by examining sclera, skin, nail bed, under the tongue, soft palate. Its duration, progression, persistent or intermittent, painful jaundice (in biliary stone) or painless jaundice (carcinoma) should be assessed. Progressive jaundice is a feature of carcinoma head of pancreas, nodes compressing porta hepatis, Klatskin tumour; intermittent jaundice is a feature of periampullary carcinoma (due to sloughing of the ampulla), stone in common bile duct. Presence of itch marks on the dorsal aspect of the hands, forearms and back suggests obstructive jaundice.

Bowel Habit

It is very important to ask history regarding proper bowel habit in chronic abdomen patients. History of diarrhoea, constipation, blood in the stool, painful defecation, tenesmus, alternate constipation and diarrhoea, clay coloured stool (seen in chronic pancreatitis, obstructive jaundice where fat is not digested due to

deficiency of pancreatic enzymes) silvery stool (seen in periampullary carcinoma where blood from the tumour necrosed area gets altered as haematin which mixes with fat). Large, loose, fatty offensive stool may be seen in chronic pancreatitis. Inflammatory bowel disease, carcinoma colon, small bowel diseases, colonic polyps, colonic tuberculosis can cause diarrhoea or diarrhoea alternating with constipation. Dark tarry coloured melaena is also typical. *Diarrhoea* is an increase in daily stool weight more than 200 gm. There may be increased stool liquidity and frequency more than 3 times per day. Stool may be semiformed. *Pseudodiarrhoea* is increased frequency without increase in stool weight which is seen in IBS, hyperthyroidism, proctitis. But for all practical purpose increased frequency may be considered as diarrhoea. Diarrhoea is called as *acute* if it lasts for 1-2 weeks; *chronic* if it is for more than 2 weeks. *Constipation* is frequency of defaecation less than 3 times a week often with hard stool or with difficulty to pass.

Appetite

Loss of appetite is an important feature of gastrointestinal malignancy whether it is stomach, small bowel, colon, and rectum. Appetite is increased in peptic ulcer. Appetite is normal in gastric ulcer but patient fears to take food due to pain. Aversion to fatty food is a feature of gallbladder disease (gallbladder dyspepsia). Loss of appetite occurs in early gastric cancers. Loss of appetite is progressive and significant in malignancy. Feeling of adequateness/*satisfaction* after meal is called as *satiety*. *Early satiety* is a feature of GI malignancy especially in carcinoma of stomach. *Anorexia* is lack of desire to eat. *Sitophobia* is fear of eating due to anticipated abdominal discomfort seen in IBS, chronic mesenteric ischaemia.

Loss of Weight

Progressive loss of weight is seen in GI carcinomas. It is also observed in pyloric stenosis. More than 10 Kg weight loss in 6 months is called as *significant weight loss* which needs proper evaluation. Often patient might not have weighed his weight at all earlier. Then it is better to ask how much muscle mass is reduced or loosening of clothes occurred. Often it is better to ask relatives about their observation of the changes in the patient earlier and now.

Fever

Abdominal tuberculosis may present with evening rise of fever. Fever may be due to malnutrition, secondary infection. Cholangitis, pancreatitis, cholecystitis, and ulcerative colitis can cause recurrent episodes of fever. Even malignancy can cause fever due to pyrogenic response or tumour necrosis.

Past History

Past history of typhoid, tuberculosis, jaundice is important. Previous history of any surgery or abdominal surgery – indication, duration of hospital stay, whether it was an emergency or elective procedure, postoperative recovery, drain placed or not, any biopsy reports revealed or not, recovery period should be noted. Long-term treatment for abdominal tuberculosis may be present. Patient might be taking drugs related to peptic ulcer for long time. Whether patient was evaluated prior to therapy or surgery by X-rays, investigations, endoscopies or not should be asked. History of blood transfusions for surgery earlier is also significant.

Personal History

History of smoking, alcohol intake, spicy food, dietary habits like regularity, interval between each food intake, type of food intake should be asked. Alcohol and smoking may lead into cirrhosis and portal hypertension; peptic ulcer disease, carcinoma, etc.

Family History

Certain gastrointestinal malignancies, ulcerative colitis, Crohn's disease often run in family.

General Examination

Anaemia, reduced weight is common in malignancy, abdominal tuberculosis. Oral cavity, teeth, jaundice, clubbing, respiration, pulse, blood pressure, skin texture, built, overall look of the patient should be checked. *Malignant cachexia* is emaciated skeletonised look seen in gastrointestinal malignancy. Built is normal in duodenal ulcer; poor in gastric ulcer; emaciated in pyloric stenosis or carcinoma.

Local Examination

Abdominal examination is essential part of chronic abdominal conditions. Examination is done in patient

in supine position in the bed exposing the abdomen from upper chest to knee level. Both hands should be on the sides of the patient. It is better to ask the patient to turn to one side (towards left as examination is always done from right side always) breathe comfortably and relax. It is also important to explain the patient about what you are going to examine. Often consent may be needed. When a male doctor examines a female patient, it is better to have a female nursing staff beside the doctor. Good day light is needed to examine the patient. It is ideal to have some conversation with the patient while examining to ease and relax the patient. Legs may be slightly flexed at knee joints (**Fig. 20.4**).



Fig. 20.4: Examination of abdomen is done with proper exposure.

Inspection

Inspection is done from the right side, foot end side

and often from head end side of the patient with eyes keeping at the level of the abdomen (**Figs 20.5A and B**).

Movements with Respiration

Localised limitation of movement with respiration can occur in localised inflammation. In peritonitis movement with respiration of the abdomen is absent. Patient will have more thoracic type of respiration.

Skin Over the Abdomen

Skin over the abdomen is looked for scar, dilated veins, redness, and oedema. Dilated veins are looked for in standing position. Dilated veins around the umbilicus with normal pattern of flow (away from the umbilicus) are due to portal hypertension – *caput medusae*. Normally above the umbilicus blood flow is upwards (to SVC) and below the umbilicus downwards (to IVC). In IVC obstruction it is upwards; in SVC obstruction it is from above downwards. Two fingers are kept very close over the vein, with pressure fingers are swept away to empty the vein, one finger is released and flow is observed; later if it is empty other finger is also released to see the flow. It is repeated again to confirm the flow in opposite direction. Scar, its length, width, margin, linear or wide scar should be checked. Linear scar means wound has healed by primary intention.



Figs 20.5A and B: Inspection of the abdomen should be done from side as well as from foot end.

Umbilicus

Umbilicus is situated normally in midway between xiphoid process and pubic symphysis. It is displaced downwards in ascites; upwards in pelvic mass – *Tanyol's sign*. It is everted in ascites, umbilical hernia; drawn in obesity; pushed towards opposite side by one sided mass (Normal equidistant line from anterior superior iliac spine to umbilicus is deviated to one side). Vertical slit in umbilicus occurs in ovarian tumour; horizontal slit in ascites.

Shape of the Abdomen

Normally abdomen is flat or only slightly scaphoid; neither full nor retracted. It may be scaphoid in thin people/starvation/advanced malignancy. Fat, fluid, flatus, faeces, and foetus cause symmetrical distension. Distension due to obesity causes inverted umbilicus. Umbilicus is everted in intra-abdominal causes. Localised area of fullness may be evident depending on where the cause is upper/lower abdomen. In patient with visceroptosis, lower abdomen becomes more prominent on standing (**Fig. 20.6**).



Fig. 20.6: Ascites on inspection. For dilated veins examination should be done on standing.

Visible Pulsation

Pulsation may be visible in thin individuals. It may be aortic pulsation. Aortic aneurysm causes visible pulsation. It causes expansile pulsation which is confirmed in knee elbow position or lateral position. Transmitted pulsation may be seen over a mass in front of the aorta like pseudocyst of pancreas, retroperitoneal mass, etc.

Visible Peristalsis

Visible gastric peristalsis (VGP) is located in the epigastrium; it is from left to right towards right lumbar region. It is a feature of pyloric stenosis. It can be stimulated by giving the patient to drink water (500 ml) or by rubbing the abdomen. Small intestinal peristalsis (VIP) is around the umbilicus. Peristalsis of transverse colon is from right to left, slow and periodic (**Figs 20.7A and B**).

Mass per Abdomen

Any visible mass or fullness should be inspected. Its location, size, shape, movement with respiration should be inspected.

Palpation

Palpation should be done from right side of the patient. Patient should lie down flat, breathing through mouth in relaxed state with head turned to opposite side. Palpation is done with flat of the hand using fingers; forearm should be in horizontal plane. Examiner may have to sit on a chair or lean on the patient to do a proper palpation. Poking with the fingers placed vertically over the abdomen should be avoided. Slight flexion of hips and knees help in relaxing the abdominal muscles and prevent patient from keeping it tight and rigid. Keeping a pillow under the knees may be useful. Clinician should make his examining hand warm by rubbing it with other hand. Continuous conversation with the patient during palpation is important to console and relax the patient.

Neville J Nicholson manoeuvre—Lower end of the sternum is pressed progressively using base of the palm of left hand progressively so that patient breathes through the abdomen relaxing it; right hand is used to palpate the abdomen (**Fig. 20.8**).



Figs 20.7A and B: VGP is stimulated by asking the patient to drink water or by rubbing the epigastric region.

Palpation should be started away from the location of suspected disease. Examine all other quadrants initially then examine the needed quadrant carefully. Often two hands placed one over the other may be used to palpate the patient's abdomen. Palpation should be done first in nontender area then in tender area.

Initially *palpation* is done to have a clear idea where exactly disease is suspected. Then deep palpation is



Fig. 20.8: Palpation of the abdomen.

done gently and slowly with asking the patient to undertake deep inspiration. *Deep palpation* is done using fingers of the hand placed over the abdomen pushing deep on each expiration. *Two hands method* is better for deeper palpation (**Figs 20.9A and B**).



Figs 20.9A and B: Method of deep palpation of the abdomen.



Fig. 20.10: Palpation in child/children is done using child's hand.

In children *child's hand* is placed over the abdomen and examiner's hand is placed over the child's hand and is palpated. When pain is present child withdraws the hand (due to tenderness) (**Fig. 20.10**).

Head raising or leg raising test (Carnett's) is done to confirm any mass if present is intra-abdominal or not. If mass becomes less prominent during these tests it is intra-abdominal; if mass becomes more prominent it means it is in the abdominal wall (**Figs 20.11 and 20.12**).



Fig. 20.11: Head raising test.



Fig. 20.12: Carnett's leg raising test. Both legs are raised above with knees straight and abdomen is inspected and palpated.

Tenderness

While palpating it is checked by looking at face of the patient and feel of the abdomen. When tenderness is mild patient tolerates but winces; when it is moderate patient winces and tightens the abdomen; when it is severe patient winces and makes abdomen rigid and does not allow further palpation.

Deep tenderness is elicited with one finger. Point of maximum tenderness (tender spot) should be elicited. In duodenal ulcer it is in transpyloric plane 4 cm right of the umbilicus. In cholecystitis tender point is below the right costal margin over the lateral margin of the right rectus muscle. Tenderness elicited here is from the fundus of gallbladder. *Murphy's sign* for chronic cholecystitis is elicited in sitting position. Patient lifts his right arm above the shoulder; examiner stands on right side of the patient and right hand fingers (or left thumb) are placed and hooked under right costal margin lateral to the right rectus muscle. When patient is asked to take deep breath, patient winces with pain during Zenith of inspiration as the inflamed gallbladder descends during inspiration and touches the examiner's fingers. Same tenderness if elicited in lying down position, it is called *Moynihan's method/sign*. Cartilage of 8th rib will be tender in cholecystitis (**Figs 20.13 and 20.14**).

Fluid Thrill

Fluid thrill is elicited when large amount of fluid (> 2000 ml; fluid under tension) is present in the peritoneal cavity (ascites). Patient's or assistant's hand is placed vertically in the midline of abdomen pressing deeply to prevent the formation of transmitted wave towards opposite side along the subcutaneous plane and also to increase fluid tension inside the peritoneal



Figs 20.13A and B: Murphy's sign is elicited in sitting position using examiner's left thumb or right hand fingers. It is done in chronic cholecystitis.



Fig. 20.14: Moynihan's test is done in lying down position for chronic cholecystitis.

cavity. One side abdomen is tapped with fingers; fluid thrill is felt with the other hand on other side. Often such fluid thrill is positive in large ovarian cyst also. But it can be differentiated by shifting dullness and Blaxland ruler test. Shifting dullness is done during percussion. Ascites may be due to congestive cardiac failure, portal hypertension, abdominal tuberculosis,



Fig. 20.15: Demonstration of fluid thrill.

peritoneal carcinoma, advanced malignancies (**Fig. 20.15**).

Blaxland Ruler Test

Urinary bladder is emptied. A flat ruler is laid over the abdomen just above the anterior superior iliac spines. With the fingers of the both hands the ruler is pushed firmly and steadily towards lumbar spine. Abdominal aortic pulsation is felt in ovarian cyst. It is not felt in ascites (**Figs 20.16A and B**).



Figs 20.16A and B: Blaxland ruler test. First ascertain the line of anterior superior iliac spine.

Dipping Method

When there is large quantity of fluid in the abdominal cavity palpation of different organs is done by dipping the fingers by which fluid is displaced away from the place.

Palpation of Different Organs

Stomach

Normally stomach is not felt on palpation. It is felt as dilated in pyloric stenosis due to chronic duodenal ulcer (cicatrised), pyloric growth. Visible gastric peristalsis (VGP), positive succussion splash, positive auscultopercussion test is significant. Dilated stomach will be below the level of umbilicus (greater curvature). VGP may be absent if gastric paresis occurs due to atony of stomach wall. Stomach mass is usually due to carcinoma, occasionally leiomyoma or sarcoma. Mass moves with respiration, freely mobile, all borders well made out, irregular surface, hard in consistency,

resonant or impaired resonant on percussion. It becomes immobile once it gets fixed posteriorly. Absence of stomach mass will not exclude carcinoma of stomach.

Liver

In infants it is palpable upto 3 years. In adult it is usually not palpable. Any palpable liver is considered as pathological. Liver is palpated using right hand. Palpation should begin well below from right iliac fossa otherwise it may be missed. Right fingers are laid flat with outer margin of the index finger held facing upwards and inwards. Fingers are pointed towards left axilla parallel to right costal margin. During deep inspiration fingers are pressed firmly to feel; during expiration fingers are moved upwards towards right costal margin. During full inspiration as fingers are moving upwards, lower margin of descended liver will come and touch the outer edge of the index finger. The fingers are kept there for further confirmation of the liver and also other features of the liver like—presence of tenderness or not; extent in cms or fingerbreadth below right costal margin; edge type—sharp or rounded; surface—smooth, irregular, granular, nodular, umbilications; consistency – soft, firm, hard. Smooth tender liver may be in amoebic liver abscess or viral hepatitis. Nodular hard liver is a feature of secondaries in liver. Umbilication is seen in liver secondaries due to central necrosis. Hepatocellular carcinoma may be smooth/nodular; soft/firm or hard. Soft, smooth liver is felt in congestive conditions. In obstructive jaundice, liver may be enlarged due to obstruction causing dilated intrabiliary radicals – *hydrohepatosis* where liver is soft and smooth; or it may be due to secondaries (nodular, hard) from carcinoma of head or periampullary region.

Spleen

Spleen is only occasionally palpable in normal person (1-3% of normal people – in New Guinea commonly). Normal spleen is 12 × 7 cm in size. It is enlarged if it is more than 14 cm. Spleen should get enlarged three times to become palpable. *Method 1:* Right hand fingers are used to palpate the spleen from right iliac fossa. Index finger is placed like palpation for liver. Fingers are moved towards left hypochondrium and

upwards in each phase of respiration. Spleen is palpated under the tip of 10th rib. *Method 2:* Left hand may be kept under the left lower chest wall and skin is moved downwards so that more lax skin is available to insinuate the right hand fingers under left costal margin. *Method 3:* While right hand is palpating, left hand is placed under the left rib cage to lift it upwards so that spleen comes forward to facilitate the palpation by right hand. *Method 4:* Spleen can be palpated from above—left side of the patient with two hands arching below the left costal margin, and during phases of respiration spleen will come down and touch the examiner's fingers. Often *tilting the patient with left side up* during palpation makes spleen to be palpated in easier way. *Method 5: Hook sign:* Hooking the left costal margin with fingers is not possible in splenic enlargement. *Method 6: Middleton's manoeuvre:* Examiner stands on left side of the patient facing towards foot end, keeps his left hand fingers hooked under left costal margin and exerts pressure over the posterolateral aspect of the lower thorax using his right hand and spleen is felt at the end of deep inspiration (**Figs 20.17A and B**).

Causes of splenomegaly—Congestive cardiac failure, malaria, portal hypertension, haemolytic anaemias, idiopathic thrombocytopenic purpura, kala-azar, lymphomas, chronic myeloid leukaemia (massive spleen), polycythaemia rubra vera, sarcoidosis, myelofibrosis, typhoid fever, autoimmune diseases, splenic abscess, splenic cyst, tuberculosis.

Hypersplenism is overactivity of the splenic function which has nothing to do with splenic size with typical features of—splenomegaly; pancytopenia; hypercellular or normal bone marrow; reversible by splenectomy. Causes for hypersplenism are—lymphoma, cirrhosis, myeloproliferative diseases, and connective tissue diseases.

Gallbladder: Gallbladder when enlarged is visible on inspection in the right hypochondrium as globular mass directed downwards and forwards below right costal margin or below the lower margin of the palpable liver just lateral to the lateral border of the right rectus muscle along the tip of the 9th rib. Mass moves with respiration; mobile horizontally, dull on percussion, soft, smooth. It may be tender if it is empyema gallbladder otherwise



Figs 20.17A and B: Palpation of spleen—different methods.

it is nontender. It is enlarged in mucocele of gallbladder, in carcinoma head of pancreas or periampullary carcinoma. It is hard in carcinoma of gallbladder.

Pancreas: It is being a retroperitoneal organ is felt if at all on deep palpation only. It is felt if there is pseudocyst of pancreas or cystadenocarcinoma or cystadenoma of pancreas. Carcinoma of head of pancreas is usually not palpable (gallbladder is palpable

here with obstructive jaundice). Pancreas in chronic pancreatitis or pancreatic cyst is better felt in lateral position from left side with patient turned towards right side with hip and knees flexed. Left subcostal and epigastric regions are deeply palpated. In this position bowel in front will be displaced making pancreas better palpable. Tenderness elicited in this position in chronic pancreatitis is called as *Mallet – Guy's sign* (**Fig. 20.18**).



Fig. 20.18: Eliciting Mallet – Guy sign in chronic pancreatitis.

Colon

Faecal mass may be felt like a colonic mass. Faecal mass yields/moulds (indents) on pressure. It subsides or reduces in size after giving enema. Distended caecum is better seen than felt as fullness in the right iliac fossa. Colonic mass is located along the anatomical line of the colon depending on the site of pathology. It is mobile but does not move with respiration, nodular, hard well localised mass. Anaemia, diarrhoea, constipation, distension are other features.

Kidney

Kidney is palpated by—bimanual palpation; ballotability. In sitting position renal angle tenderness should be checked (**Figs 20.19 and 20.20A to C**).

Abdominal girth measurement is done at umbilical level. Periodic measurement is done to assess the progress of the disease.

Percussion

Liver dullness is elicited in the 5th intercostal space in midclavicular line on right side. Liver span is assessed. It is 12-15 cm in adult. Percussion is started



Fig. 20.19: Renal angle tenderness is elicited in sitting position.

from right 4th space downwards until dullness is reached and continued upto the lower margin of the dullness. Liver dullness is reduced in severe emphysema, right sided pneumothorax. It is obliterated in perforation of viscus causing gas under diaphragm.

Percussion over the mass is important to locate the anatomical plane. Abdominal wall masses, masses in front of the bowel like liver, spleen, gallbladder are dull on percussion. Mass arising from bowel is impaired resonant. Retroperitoneal mass is resonant (as bowel is present in front) on percussion like pancreatic mass, renal mass, aortic aneurysm, para-aortic lymph node mass, retroperitoneal tumours or cyst. Mass in the upper abdomen when is dull during percussion should confirm whether it is continuous with liver dullness or not.

Shifting dullness for free fluid should be checked (1000 ml of fluid should be present). **Puddle sign** is assessing small quantity of free fluid in the abdominal cavity in knee elbow position (120 ml). It is checked in same position often by auscultopercussion also.



Figs 20.20A and C: Different methods of kidney palpation.

Note: Proper US may detect 30 ml of fluid. *Grading of ascites:* Grade 1—Detectable only by careful examination; Grade 2—Easily detectable small volume; Grade 3—Obvious ascites but not tense; Grade 4 — Tense ascites.

In ascites, it is dull in the flanks but resonant on the summit—centre whereas in ovarian cyst it is resonant in periphery but dull in the centre.

Traube's area: It is bounded above by lung resonance; below by left costal margin; on the right side by left border of the liver and on the left side normal splenic dullness. It lies in left lower chest behind 9th, 10th and 11th ribs. Normally it is resonant as it is occupied by stomach. It becomes dull in left sided pleural effusion, *splenomegaly*, and *stomach (fundus) with solid tumour or fluid*, enlarged left lobe of the liver, massive pericardial effusion. It is shifted upwards in left lower lobe collapse/left lung fibrosis/left side diaphragm paralysis.

Percussion for splenic dullness: Method 1: (Nixon's)— Patient is turned towards right side (left up) and percussion is started at posterior axillary line proceeding perpendicularly towards anterior costal margin. Upper border of dullness is 8 cm above the costal margin in normal people. Dullness more than 8 cm signifies splenic enlargement. *Method 2 (Castell's):* – Resonant note normally felt while percussing in supine position along the lowest intercostal space in anterior axillary line (left) becomes *dull* on *full inspiration* in case of splenomegaly.

Percussion over the renal angle: Normally renal angle (angle of erector spinae and 12th rib) is resonant due to colon underneath. In kidney enlargement angle is occupied by enlarged kidney in deeper plane reflecting the colon in front and medially making it dull on percussion (**Fig. 20.21**).

Auscultation

Bowel sounds: Normal bowel sounds (Borborygmi) are 2-4 in number per minute. If it is more than 5 it is *hyperperistaltic*—feature of early obstruction, enteritis, carcinoid syndrome. Absence bowel sounds is called as *silent abdomen*. It is observed in paralytic ileus, late intestinal obstruction, acute peritonitis, acute pancreatitis, acute mesenteric ischaemia. In late



Fig. 20.21: Percussion over the renal angle in sitting position. It is normally resonant.

paralytic ileus, high pitched tinkling sound is heard due to spill over of contents from one loop to another – ‘bells at evening pealing’. Bowel sounds are checked with bell of the stethoscope in umbilical region. It is small bowel peristalsis which is heard.

Bruit around umbilicus may be due to renal artery stenosis. *Bruit* above the umbilicus may be due to aortic aneurysm.

Bruit over liver may be due to increased vascularity – haemangioma, HCC, hepatic artery aneurysm. Hepatic rub suggests perihepatitis.

Kenawy's sign: By placing stethoscope beneath the xiphoid process, a venous hum is heard in portal hypertension which is louder during inspiration. It is due to engorgement of the splenic vein and during inspiration spleen is compressed making it louder.

Cruveilhier-Baumgarten syndrome is venous hum heard between xiphisternum and umbilicus in portal hypertension due to patent congenital umbilical vein draining portal vein.

Splenic rub suggests splenic infarction, chronic myeloid leukaemia, endocarditis, sickle cell disease. *Bruit* in this region may be due to splenic artery aneurysm.

Auscultopercussion test for stomach dilatation: Stethoscope is placed over the xiphisternum. With the finger, gentle strokings are done from epigastrium,

adjacent to stethoscope, placed outwards towards left side. It is done repeatedly from above downwards left side. Change in the sound at the margin of greater curvature will be obvious. All points from above downwards are joined to mark the greater curvature of the stomach. It is above the level of the umbilicus in normal individual. It shifts below in gastric outlet obstruction like pyloric stenosis, carcinoma pylorus. Only greater curvature is assessed as dilatation takes place at greater curvature.

Succussion splash: Stethoscope is placed over the epigastrium. Using thumb and fingers of both hands, which are placed on each side of lower chest wall, patient is held firmly and shaken to hear splashing sounds of fluid in the stomach. Patient should not take any fluid for at least 4 hours as succussion splash is heard even in normal person giving false positive result. Positive succussion splash suggests gastric outlet obstruction.

Examination of Left Supraclavicular Lymph Nodes

It is enlarged when there is spread from gastrointestinal malignancies through thoracic duct. It is located deep to deep fascia between two heads of the sternocleidomastoid muscle. This Virchow's node enlargement is called as *Troisier's sign*. It suggests advanced malignancy. FNAC of this node will give the histological diagnosis of adenocarcinoma. It is felt using finger dipping deep between two heads of sternomastoid muscle (**Fig. 20.22**).



Fig. 20.22: Palpation of left supraclavicular lymph node – Troisier's sign.

Digital Examination of Rectum

It is done to look for secondaries (Blumer shelf) in anterior aspect above the prostate level. It is nodular hard with free mucosa (not adherent). It suggests advanced malignancy. It is due to peritoneal spread. It is useful in Crohn's disease (fissure in ano or fistula in ano), and other chronic conditions.

Pervaginal examination is a must in females especially in lower abdomen masses and when *Krukenberg* tumour (Carcinoma stomach) of ovary is suspected.

Systemic Examinations

Examination of respiratory system, spine (for secondaries or tuberculosis), central nervous system is essential (**Figs 20.23 and 20.24**).



Fig. 20.23: Psoas spasm causing hip in flexion due to psoas abscess.



Fig. 20.24: Examination of spine is essential.

Investigations

Detailed investigations and discussions of different conditions are beyond the scope of this book. Students should refer *SRBs Manual of Surgery, 3rd edn* and *Bedside Clinics in Surgery* for in detail discussions.

Blood – Hb%, ESR, relevant tumour markers.

Stool examination: Occult blood, steatorrhea (fat), creatorrhea (muscle fibres seen in chronic pancreatitis), mucous in stool, microscopy, culture.

Gastric Function Tests

Patient is overnight fasting; should not take any antacids and anticholinergics for 24 hours. Nasogastric tube is passed early morning and gastric juice is aspirated under fluoroscopy after confirming the tip of tube is at mid stomach level. Normally it is around 70 ml. If it is more it suggests pyloric stenosis or hypersecretion. Tube now is connected to low pressure 5 cm Hg suction to have continuous aspiration. Later one hour aspiration is collected. It is called as *morning basal secretion*. Free acid level means only HCl level; total acid level means HCl plus other acids in the stomach.

Dragstedt test: Gastric content is aspirated through continuous low pressure suction for 12 hours from 9. PM to 9. AM and the juice is collected. Normal volume is 400 ml. It is increased in duodenal ulcer due to vagal hyperactivity; increased very much more than a litre in Zollinger-Ellison syndrome. Normal HCl level in this is 10–20 mEq. It is 40–80 mEq in duodenal ulcer (↑); 100–300 mEq in ZE syndrome (↑↑); 5–15 mEq in gastric ulcer (↓). Basal secretion is the secretion from the parietal cell mass *in resting* condition. It is < 5 mEq/hour normally; > 5 mEq in duodenal ulcer; 1–2 mEq in gastric ulcer. Peak/maximal secretion is secretion in one hour after stimulation. Stimulation may be using pentagastrin (now used)/ Kay's augmented histamine/Hollander's insulin. It gives *maximal/ peak acid output*.

Pentagastrin test: Initial basal secretion is collected. 6 µ gm of pentagastrin is injected IM/SC and 15 minutes gastric samples are taken for one hour. Peak/maximum acid output is assessed. Normal is 25–27 mEq; in gastric ulcer it is upto 15 mEq; in duodenal ulcer it is 35–38 mEq; in ZE syndrome it is > 60 mEq/very high; in carcinoma stomach it is very low.

Kay's augmented histamine test: After collection of basal fasting sample, mepyramine maleate 100 mg IM is injected to neutralise histamine side effects without interfering gastric effects. After 30 minutes, histamine acid phosphate 0.04 mg/kg is injected S/C. Free acid level (only HCl) is assessed. In stomal ulcer it is 30-35 mEq; in duodenal ulcer it is 30-40 mEq; in gastric ulcer it is less than 15 mEq.

Hollander's insulin test: It is useful to assess post-operatively and confirm the completeness of vagotomy. 0.2 units/kg wt of insulin is injected IV to a fasting patient so as to create hypoglycaemia below 35 mg% which stimulates the parietal cells through hypothalamus and vagus causing acid secretion. Patient with complete vagotomy will not show the increased acid level. Increase in acid secretion in first one hour is called as *early response* and if it is more than 30 mmols/L suggests incomplete vagotomy. *Delayed response* is between 1st and 2nd hours and is due to delayed gastrin release.

Chew and spit test: Nasogastric tube is passed. Patient chews a meal and spits it out to stimulate acid production through vagus. Gastric contents are aspirated and studied for acid level.

Barium/Contrast Studies

Barium meal X-ray: Barium meal X-ray is done using barium sulphate (95% w/v) solution of which 400–600 ml is given orally. Gastrograffin is also often used. It is done in empty stomach. Microcrystallised barium sulphate solution is better. Procedure should be done under fluoroscopic guidance. Buscopan injection is given to the patient to delay the gastric emptying. Glucagon also can be used. Effervescent tablet (calcium carbonate and antifoaming agent) is given to the patient. 200 ml of barium sulphate solution is given to drink. X-rays are taken to get *double contrast* barium meal X-rays. Indications for barium meal X-ray—*Duodenal ulcer*—shows absent/deformed duodenal cap; *Benign gastric ulcer*—shows niche (due to ulcer) and notch (due to spasm); Gastric outlet obstruction; *Carcinoma stomach*—irregular filling defect; *Carcinoma head of pancreas*—pad sign; *Periampullary carcinoma*—Frostberg reverse '3' sign; *Chronic duodenal ileus*—obstruction at mid 3rd part of the duodenum; Stomal ulcer—ulcer crater at stoma;

Duodenal diverticula—trifoliate duodenum; Trichobezoars; Gastric fistulas; *Pseudocyst of pancreas*—widened vertebrogastric angle.

Barium meal follow through X-ray is done as late films often after giving prokinetic agents like metoclopramide.

Enteroclysis (small bowel enema): It is visualisation of entire length of small intestine—to assess anatomical problems. Indications are small bowel diseases/ileocaecal tuberculosis, stricture, small bowel tumours, partial obstruction and Crohn's disease. **Technique**—Patient is prepared overnight with empty stomach and laxatives. Nasojejunal tube is passed. Prokinetic drug like metoclopramide is given. Microbarium sulphate solution (50% w/v) or gastrograffin or water soluble iodine dye solution is (500–800 ml) passed through the tube. Under fluoroscopic guidance X-rays are taken as required. Features such as narrowing, smooth/irregular filling defect, localised dilatation, obstruction or features of specific conditions are looked for. In conditions like ileocaecal tuberculosis enteroclysis and barium enema X-rays are combined. Problems with enteroclysis are poor patient acceptance, and technical difficulty. *Capsule endoscopy* or enteroscopes are better options to visualise the small bowel. When nasojejunal tube is not able to be passed *barium meal follow through X-ray* is done by taking late films of barium meal (**Figs 20.25 to 20.28**).



Fig. 20.25: Barium meal X-ray showing polypoid growth.

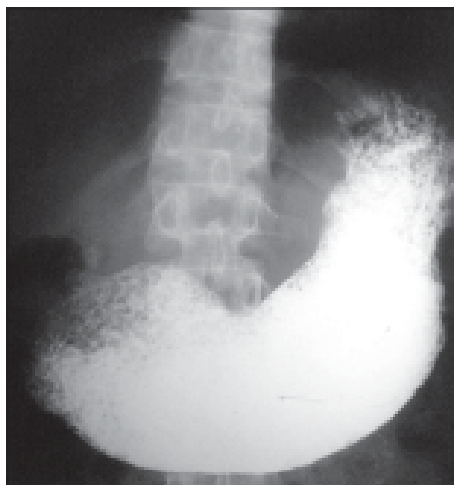


Fig. 20.26: Barium meal X-ray showing gastric outlet obstruction.

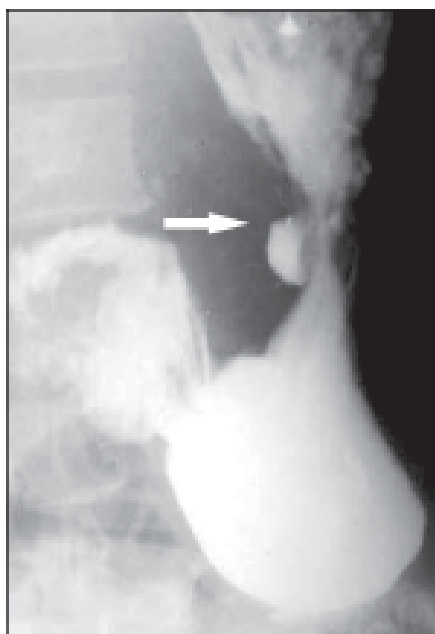


Fig. 20.27: Benign gastric ulcer showing Niche on the lesser curve.

Barium Enema X-ray

Technique of barium enema X-ray—24 hours liquid diet, laxatives for two nights and enema on previous night are the required preparations for barium enema X-ray. About one litre of barium sulphate/microbarium sulphate solution (25% w/v) is infused per anally into



Fig. 20.28: Trifoliate duodenum—late effect of chronic duodenal ulcer as a secondary diverticula.

the colorectum using a enema tube from an enema can. Patient will be initially in left lateral position and later in prone position. In children a Foley's catheter with inflation is used to maintain the retention of enema. Procedure is often observed under fluoroscopy. Injection buscopan is injected (20 mg IV) to relax the colon. X-ray film is taken after *complete filling*. Patient is asked to evacuate the barium and later post-evacuation film is taken. Air is insufflated into the colon to get air contrast film. Additional different view films are taken to see the suspected area properly.

Indications for barium enema are carcinoma colon; Ileocaecal tuberculosis [combined with Enteroclysis;] Ulcerative colitis; Crohn's disease; Ischaemic colitis; Colonic polyps; Intussusception; Congenital megacolon; Gastrojejunal fistula; Congenital diaphragmatic hernia (Bochdalek).

Different findings: *a. Hirschsprung's disease:* Barium enema is done to look for the extent of disease and three zones. Foley's catheter *should not be used* while doing barium enema in case of Hirschsprung's disease. Here barium in dilute saline is used – not in water.

1. *Distal immobile spastic segment*, i.e. aganglionic zone.
2. A proximal, *middle transitional zone* of about 1-5 cm length with less, sparse number of ganglions (*Cone*).
3. A still more proximal, *hypertrophied dilated segment* is actually the normal ganglionic area.

b. Carcinoma colon: Irregular filling defect; Apple core lesion especially on left side; Metachronous growths (growths in different parts of the colon) should be looked for – 5% common; Narrowing – left sided lesion. *c. Ulcerative colitis:* Loss of haustrations; Contracted smooth colon; Presence of pseudopolyps; *Collar button ulcers*—contiguous mucosal involvement; *Hose pipe/pipe stem* lesions; Increased presacral space more than normal (normal is < 1 cm); Reflux ileitis; Rectum is almost always involved. *d. Ileocaecal tuberculosis* - Pulled up caecum due to fibrosis and contraction; *Obtuse ileo caecal angle* (normal angle is acute); Hurrying of barium due to rapid flow—*Stierlin sign*; Narrow ileum with thickened ileocaecal valve, *Fleischner-inverted umbrella sign*; Incompetent ileocaecal valve; Ulcers and strictures in terminal ileum—*napkin* lesions; *Gooseneck appearance*—ileum hanging from fibrosed; pulled up caecum. *e. Crohn's disease:* Aphthoid ulceration; Skip lesions; Rectum is not commonly involved; *String sign of Kantor*; *Cobble stone appearance*—pseudosacculations; *Raspberry/rose thorn* appearance; Fistula or strictures. *f. Sigmoid diverticula:* *Saw teeth appearance* of sigmoid colon—concertina like—serrated appearance; *Champagne glass sign*—partial filling of barium with stercolith inside the diverticula; Fistula to adjacent structures. *g. Intussusception* - *Claw sign* – *coiled spring sign*: pincer end; Empty right iliac fossa—mainly in plain X-ray abdomen with multiple air fluid levels (on ultrasound – *target sign/pseudokidney sign/bull's eye sign*). *h. Ischaemic colitis:* *Thumb print sign* in splenic flexure.

Other signs in barium enema X-ray: *Stacked coin appearance* due to submucosal haemorrhages in Henoch Schonlein purpura; Scalloped edges in colon in pneumatosis intestinalis (**Figs 20.29 and 20.30**).

Hypotonic duodenography: It is same as barium meal X-ray but duodenal hypotonia is achieved by giving injection glucagon or buscopan so that air contrast hypotonic barium X-ray study shows better pictures.

Oral Cholecystography (OCG, Graham-Cole Test)

Patient is advised to have fat free diet for 3 days. Previous night 6 tablets of *iopanoic acid* (Telepaque) is given orally. Next morning plain X-ray abdomen is taken to visualise the gallbladder. Later fatty meal

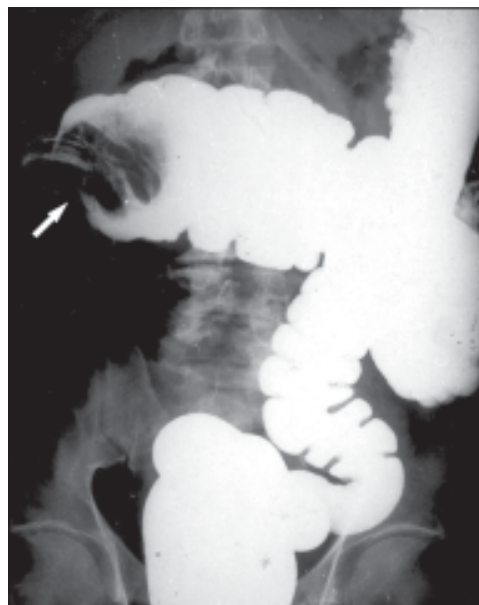


Fig. 20.29: Barium enema X-ray showing typical claw sign/coiled spring sign – in transverse colon – ileocolic type – commonest type.

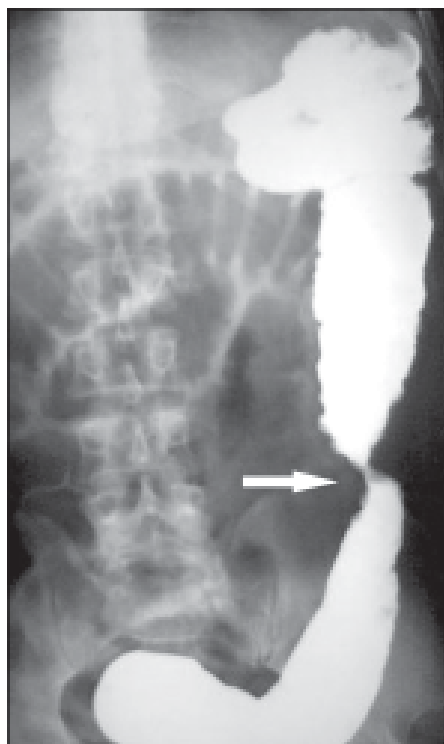


Fig. 20.30: Barium enema X-ray showing growth with stricture in the descending colon.

is given and X-rays are taken at 10, 15, 30 and 60 minutes to see the change in the size of the gallbladder (which should be less in size compared to the earlier film, as the gallbladder contracts on stimulation if it is functioning normally) (**Fig. 20.31**). Smooth filling defect signifies non-opaque stone.



Fig. 20.31: Oral cholecystogram done to see the function of the gallbladder.

Contraindications: Patients with serum bilirubin > 3 mg%, acute cholecystitis. OCG is not done now.

Intravenous cholangiography: It is done to visualise bile ducts and biliary tree, by injecting IV *Meglumine ioglycamate* (Biligram) and taking X-Ray abdomen. It can be combined with OCG. Problems with this method are poor visualisation, drug reaction. It is not very useful if serum bilirubin is > 3 mg%.

Endoscopic Retrograde Cholangio-pancreatography (ERCP)

Through a side viewing gastroduodenoscope, sphincter of Oddi is cannulated, dye is injected and biliary and pancreatic tree is visualised. It is done under C-ARM guidance. It is done under sedation like midazolam

or using propofol anaesthesia. Patient is placed in prone position with the head turned towards right. After passing gastroduodenoscope, sphincter is identified and cannulated. Under visualisation 3 ml of water soluble iodine contrast is injected into the bile duct and pancreatic duct. When cannula goes upwards beside vertebra, it is in bile duct; and if cannula goes across the vertebra it is in pancreatic duct.

Indications—Malignancy—irregular filling defect; Chronic pancreatitis—*chain-of-lakes* appearance; Congenital anomalies, stones (**Fig. 20.32**); Stricture of biliary tree; Choledochal cyst; For sampling of biliary and pancreatic juices for analysis and cytology; Brush biopsy from tumour site.



Fig. 20.32: ERCP showing stone in common bile duct.

Therapeutic uses—Extraction of biliary duct stone; Nasobiliary drainage; Stenting of tumour in the CBD or in the pancreas; Dilatation of the biliary stricture; Endoscopic papillotomy.

Complications – Pancreatitis; Duodenal injury; Cholangitis; Bleeding.

Relative contraindications—Acute pancreatitis; Previous gastrectomy; Altered prothrombin time

(corrected by injection Vitamin K, FFP); Bleeding disorders.

Percutaneous Transhepatic Cholangiography (PTC)

It is done in case of severe obstructive jaundice under coverage of appropriate antibiotics and after control of any bleeding tendency. With the help of fluoroscopy, *Chiba (university) or Okuda needle* which is long, flexible, thin, blunt, without beveled end, is passed into the liver through right 8th intercostal space in midaxillary line. Once needle is in the dilated biliary radicle, bile is aspirated (sent for culture, cytology, analysis); and then water soluble iodine dye is injected into the same so as to visualise the dilated biliary radicles, also the site and extent of any obstruction (i.e. tumour, stricture). Procedure can be used for therapeutic stenting across the biliary tree through any obstruction either in the hepatic ducts or in the CBD into the duodenum. **Complications:** Bleeding, biliary leak, biliary peritonitis and septicaemia.

Magnetic Resonance Cholangio-pancreatography (MRCP)

MRCP is a non-contrast imaging method, better than ERCP as diagnostic tool in biliary and pancreatic diseases. T2 W1 images are used.

Gastroscopy

It is visualisation of interior of stomach, duodenum and oesophagus. *It is used for diagnosing any pathology*—Gastric ulcer; duodenal ulcer; gastritis; stomal ulcer; carcinoma stomach; oesophagitis; oesophageal varices. Biopsies from the suspected cases of malignancy or for *Helicobacter pylori* can be taken. *Endosonography* can be done to assess the staging, operability of carcinoma stomach or oesophagus. Fibreoptic flexible gastroduodenoscopy also can be used. *Videoendoscopy* is used not only for diagnosis but also mainly for therapeutic procedures. Both end viewing and side viewing gastroscopes are available. For therapeutic procedures and ERCP, side viewing gastroscopes are required.

Therapeutic procedures: Variceal injection or ligation; Stenting of pseudocyst of pancreas through gastro-

scopy; polyp removal; submucosal resection; for ERCP—diagnostic and therapeutic procedures.

Procedure: Gastroscopy is done following eight hours of fasting. After lignocaine spray into the oral cavity; gastroscope is passed gently down the oesophagus when the patient does the swallowing action. Once the scope is inside the stomach, air is inflated and different parts of the stomach are visualised. Fundus is visualised by retropulsion. Scope is passed through the pylorus to see 1st and 2nd parts of the duodenum. Looked for any pathology and if required biopsy is taken (**Figs 20.33 and 20.34**). Often midazolam sedation is beneficial to have an easy passage.

Complications: Bleeding, aspiration, perforation (rarely).



Fig. 20.33: Gastroscopy is being done.

US Abdomen

It is very useful to identify gallstones, liver pathology. But not a good method for assessing the pancreas.

Radioisotope Scanning

Inorganic iodide is given orally 2 days prior to block the thyroid. I^{131} labelled serum albumin or ^{99m}Tc Technetium is administered. 10 minutes later scanning is done in supine position. It is useful to find out focal and diffuse diseases; abscess like in

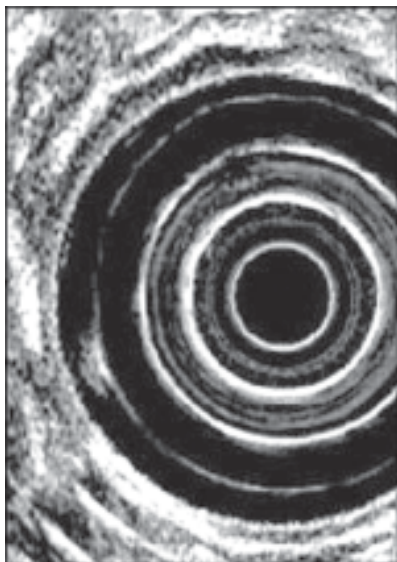


Fig. 20.34: Endosonography of stomach.

liver or subphrenic space; inflammatory pathology. ^{75}Se -labelled methionine is used to scan pancreatic lesions. It is also useful in GI bleeding. Blood loss is measured with help of RBCs labeled with Cr^{51} which is injected intravenously.

Selective angiography of superior mesenteric artery and inferior mesenteric artery is often helpful in detecting the site of bleed. Once identified, therapeutic embolisation can be used to control the bleeding. Bleeding of 0.5 ml/minute can be detected.

Diagnostic laparoscopy; laparoscopic ultrasound are also useful.

Features of Chronic benign Gastric Ulcer

It may be due to atrophic gastritis, smoking, alcohol; Typical pain is more after taking food and is relieved by inducing vomiting. Vomiting per se as a symptom is seen in 15% cases. Appetite is normal but avoids food due to pain and so loses weight. Deep tenderness

in mid epigastrium; Periodicity, haematemesis/melaena (25%) are other features.

Complications are – hour glass contracture, tea-pot deformity, erosion into left gastric/splenic arteries, perforation and malignant transformation. Risk of carcinoma is 6-23%. Patients with gastric ulcer treated with anti-ulcer drugs like proton pump inhibitor will become symptomatically better but ulcer heals partially and retains its potentiality to transform into carcinoma stomach. Giant gastric ulcer is benign gastric ulcer more than 3 cm in size. Benign ulcer occurs usually in the lesser curve whereas ulcer in greater curve is commonly malignant. Barium meal X-ray shows *niche* (ulcer crater) and *notch* on opposite side (due to spasm of circular muscle). *Lesser curve ulcer is usually benign; greater curve ulcer is commonly malignant* (Fig. 20.35).

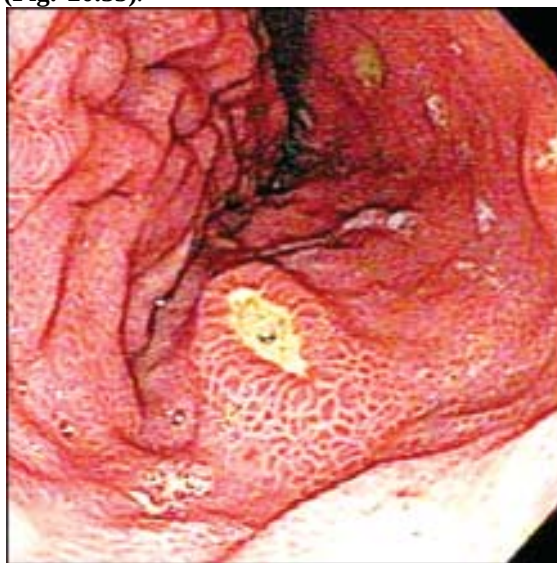


Fig. 20.35: Multiple ulcers in the stomach on gastroscopy.

Features of Duodenal Ulcer

‘Hurry, worry, curry’—stress, anxiety are the basic aetiological factors; Common in blood group O positive; *Helicobacter pylori* infection is seen in more

Types of gastric ulcer (Daintree Johnson)

Type I: In the antrum near the lesser curve	55%	Normal acid level
Type II: Proximal gastric ulcer with duodenal ulcer	25%	High acid level
Type III: Pre-pyloric ulcer	15%	High acid level
Type IV: Gastric ulcer in the proximal stomach or cardia	05%	Normal acid level

than 90% of duodenal ulcer. Other causes are NSAIDs, steroids, alcohol, smoking, hyperparathyroidism. Anterior ulcer perforates (2%); posterior ulcer bleeds (5%). Pain is typically in duodenal point. Haematemesis and melaena are more common. Patient has more appetite and eats more to relieve pain and so gains weight. Hunger pain, early morning pain, periodicity, water brash, melaena are other features. *Complications* are pyloric stenosis, bleeding, perforation and penetration into pancreas. Chronic duodenal ulcer will never turn into malignancy. Gastroscopy, biopsy for *Helicobacter pylori* is needed. Barium meal X-ray shows deformed or absent duodenal cap (**Fig. 20.36**).

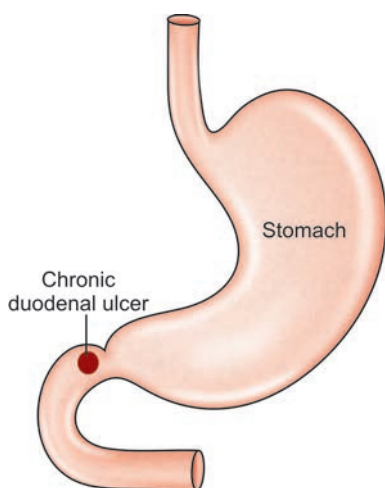


Fig. 20.36: Duodenal ulcer location.

Features of Gastric Outlet Obstruction

Pyloric stenosis is due to congenital/chronic duodenal ulcer/carcinoma pylorus. Hypochloremic, hyponatraemic, hypokalaemic, hypocalcaemic, hypomagnesaemic metabolic alkalosis with *paradoxical aciduria* is typical. Persistent pain, vomiting, decreased appetite and loss of weight, without any periodicity, visible gastric peristalsis, positive succussion splash, positive auscultopercussion test are the special features. *Barium meal X-ray shows*—Absent duodenal cap, if it is due to cicatrised chronic duodenal ulcer; Greater curvature is below the level of iliac crest; Mottled stomach due to retained food particles which gives coated/mosaic appearance; Barium will not pass into the duodenum; Dilated stomach.

Carcinoma Stomach

It is the captain of men of death. It is more common in Japan. It can be familial (Napolean family). *H. pylori* is the main causative agent. Diet (smoked fish), gastric polyp, pernicious anaemia, gastric remnant, smoking, alcohol, benign gastric ulcer, chronic gastritis are the other causes.

Clinical features: Recent onset of loss of appetite and weight; upper abdominal pain; vomiting with features of gastric outlet obstruction, i.e. (VGP+ve, auscultopercussion test, +ve succussion splash); Mass abdomen: Mass in pylorus lies above the umbilicus, nodular, hard, with impaired resonance, mobile, moves with respiration, all border well made out; Dysphagia when mass is in upper epigastrium; When it arises from the body of stomach, it may present as only mass abdomen. Along with jaundice, liver may be palpable with secondaries which are hard, nodular (50%) with umbilication. Ascites; +ve Troisier's sign (Virchow's node in neck); +ve rectovesical secondaries (Blumer shelf); +ve Trousseau sign - migrating thrombophlebitis, also seen in carcinoma pancreas; anaemia, cachexia; haematemesis, melaena; occasionally carcinoma stomach can present as *perforation* to begin with. Rarely present as secondaries in the liver with silent primary in stomach. Secondaries in umbilicus, as sister Joseph's nodules.

Classifications: *Lauren's*—intestinal, diffuse. *Japanese* (for early gastric cancer – involvement of mucosa or submucosa with or without lymph nodes)—protruded, superficial elevated or flat or depressed, excavated. *Borrmann's* (for advanced cancer—involve muscularis or serosa with or without lymph node spread) – polypoid; ulcerated with clear margin or without clear margin; diffuse; unclassified. *Leather bottle stomach (Linitis plastica)* is diffuse type of carcinoma of stomach with mother of pearl look with enormous proliferation of fibrous tissue in submucosa (**Fig. 20.37**).

Gallstones and Chronic Cholecystitis

Gallstones

Types: (1) *Cholesterol stones* are 6% common, often solitary. (2) *Mixed stones* are 90% common. It contains cholesterol, calcium salts of phosphate, carbonate,



Fig. 20.37: Gastroscope view of bleeding carcinoma of stomach.

palmitate, proteins and are multiple, faceted. (3) **Pigment stones:** are small, black or greenish black, multiple, often they can be sludge like. Common in 'Fat, Fertile, Forty, Flatulent, Female'. Common in western countries and in North East India. **Effects of the gallstones:** (a) *In the gallbladder*—Silent asymptomatic stones; Acute cholecystitis; Chronic cholecystitis; Empyema gallbladder; Perforation causing biliary peritonitis or peri cholecystitic abscess; Mucocoele of gallbladder; *Limey gallbladder*; Carcinoma gallbladder. (b) *In the CBD*—Secondary CBD stones; Cholangitis; Pancreatitis; *Mirizzi syndrome* (compression of CBD by stone from cystic duct or cholecystocholedochal fistula). (c) *In the intestine*—Cholecystoduodenal fistula causing *gall-stone ileus* and so intestinal obstruction.

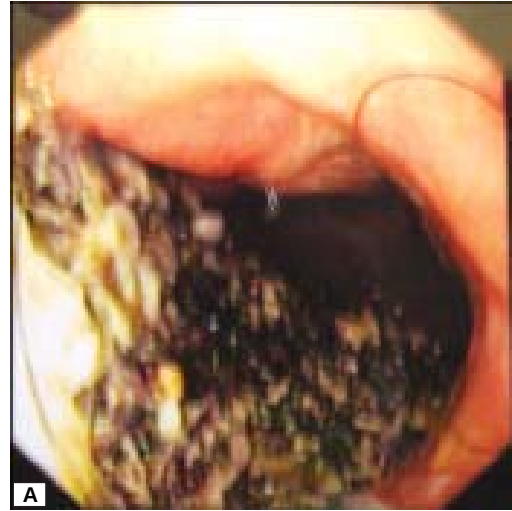
Chronic cholecystitis: It is chronically inflamed, thickened gallbladder, which is non-functioning and nondistending. **Causes:** Gallstones; Cholecystoses; Chronic acalculous cholecystitis.

Clinical features: 1. Pain in right hypochondrium may be colicky, or persistent. 2. Positive *Murphy's sign* where, in sitting position during deep inspiration, patient winces with pain at the summit of the respiration while palpating in right hypochondrium. 3. Flatulent dyspepsia.

Trichobezoar

It is hair-ball commonly seen in a stomach of *females with psychiatric illness* who swallow hair regularly. It forms a ball like mass occupying the full stomach (**Figs 20.38A and B**).

Clinical features: Haematemesis; Gastritis; Loss of appetite; Perforation; occasionally mass in the epigastrium which can be molded. Barium meal is confirmative.



A



B

Figs 20.38A and B: Trichobezoar—endoscopic view and gross after extraction (Courtesy Dr BM Nayak, Professor in Surgery, KMC Mangalore).

Chronic Pancreatitis

It is persistent progressive irreversible damage of the pancreas due to chronic inflammation. It can be chronic relapsing pancreatitis or chronic pancreatitis. It can be chronic non-calcifying or calcifying pancreatitis. Stones may be in the duct or in the parenchyma. Chronic pancreatitis is more common in males, common in Kerala South India (Induced by diet rich in Tapioca).

Aetiology: Alcohol; stones in biliary tree; malnutrition, diet; hyperparathyroidism; hereditary (Familial hereditary pancreatitis); idiopathic; trauma; congenital anomaly, etc. (Pancreatic divisum).

Pathology: It shows atrophy of acini, hyperplasia of duct epithelium, interlobular fibrosis, calcifications, ductal dilatation, with strictures in the duct.

Clinical features: (1) *Pain in epigastric region*, persistent and severe, which radiates to back. This pain is due to irritation of retropancreatic nerves, or due to ductal dilatation and stasis, or due to chronic inflammation itself. (2) *Exocrine dysfunction:* Diarrhoea, asthenia, loss of weight and appetite, steatorrhoea (signifies severe pancreatic insufficiency), malabsorption, etc. (3) *Endocrine dysfunction:* Diabetes mellitus. (4) *Mild jaundice* is due to narrowing of retropancreatic bile duct and cholangitis. (5) *Mass per abdomen*, just above the umbilicus, tender, nodular, hard, felt on deep palpation, not moving with respiration, not mobile, resonant on percussion. **Chronic pancreatitis can lead to carcinoma pancreas.**

Complications of chronic pancreatitis: Pseudocyst of pancreas; Pancreatic ascitis; CBD stricture; Duodenal stenosis; Portal or splenic vein thrombosis; Peptic ulcer; Carcinoma pancreas (Fig. 20.39).



Pancreatic Tumours

Classification

A: Exocrine tumours: *Benign:* Benign cystadenoma. It is rare. *Malignant:* (a) *Adenocarcinoma* in ampulla or periampullary region or head of pancreas. Periampullary carcinoma may consist any of the component - duodenal mucosa, CBD, pancreatic duct component or all. Occasionally squamous cell carcinoma or combination of adenosquamous can occur. (b) *Cystadenocarcinoma* of pancreas occurs commonly in body and tail of the pancreas, which usually attains a large size (5%). **B: Endocrine tumours.** **C: Lymphomas.**

1) Insulinoma	Whipple's triad
2) Gastrinoma	Peptic ulcer
3) Glucagonoma	Diabetes, necrolytic migratory erythema
4) Vipoma (Verner Morrison syndrome)	Watery diarrhoea, hypokalaemia
5) Somatostatinoma	Diabetes, steatorrhoea

Exocrine pancreatic tumours: Aetiology: Smoking; alcohol; high energy diet rich in fat; chronic pancreatitis; familial pancreatitis.

Sites: Head and neck region; Ampullary and periampullary region; Body and tail.

Pathology: (a) **75% are adenocarcinoma**, common in elderly people. Arising from primitive cells or acinar cells or duct cells. It is common in head, neck and ampullary region. Most often it begins as carcinoma in situ. (b) **Cystadenocarcinomas** occurs 1% of all pancreatic malignancies. They are large cystic tumours, which are slow growing, occurring in the body and tail of the pancreas. They are commonly papillary- cystic tumours. Occasionally mucinous cystic tumours are also seen.

Clinical features: Ampullary tumours mainly present with *jaundice and weight loss*. Carcinoma head and neck tumours present with weight loss and jaundice. Cystadenocarcinoma of pancreas present with pain, weight loss and mass.

Jaundice is of obstructive nature which is of short duration, severe, progressive, associated with pruritus (due to deposition of bile salts in the skin which releases histamine). Painless jaundice is seen in ampullary malignancies. In periampullary carcinoma necrosis of tumour occurs sometimes, as a result of which jaundice may reduce temporarily thus becoming *intermittent*.

Pain present in the right hypochondriac, epigastric, or left hypochondriac region depending on location of the tumour. Back pain when it is present is due to involvement of retropancreatic nerves, or pancreatic duct obstruction or stasis.

Diarrhoea, steatorrhoea, silvery stool (due to undigested fat mixing with metabolized blood which is derived from the ooze of periampullary growth); loss of appetite and weight; scratch marks on the back.

Migratory superficial thrombophlebitis—Trousseau's sign (10%) is due to release of platelet aggregating factors from the tumour or its necrotic material (Trousseau himself died of carcinoma pancreas who had migrating thrombophlebitis).

Ascites; Left supraclavicular palpable lymph node; secondaries in rectovesical pouch (Blumer's shelf).

Gallbladder may be palpable which is nontender, soft, globular, smooth, moving with respiration, mobile horizontally, dull on percussion. *Courvoisier law* favours gallbladder enlargement.

Liver is enlarged, smooth, firm, nontender, due to dilated bile filled biliary radicles—*Hydrohepatosis*. Liver can show multiple hard nodules due to secondaries.

Cystadenocarcinoma of pancreas can present with mass in epigastric region, which is nonmobile, not moving with respiration, smooth, soft, nontender.

Splenic vein thrombosis with splenomegaly (10%) can occur.

Hirschsprung's Disease

It is a congenital, familial condition occurring in newborn due to the *absence of ganglion cells—Auerbach's and Meissner's plexus in anorectum*, which may extend proximally either a part or full length of the colon.

Types:

1. Ultra-short segment HD—Only anal canal and terminal rectum is aganglionic.
2. Short-segment HD—Anal canal and rectum is completely involved.
3. Long-segment HD—Anal canal, rectum and part of the colon is involved.
4. Total colonic HD—Anal canal, rectum and full length of the colon is involved.

It has got three zones: 1. *Distal immobile spastic segment*, i.e. aganglionic zone; 2. A proximal, *middle transitional zone* of about 1-5 cm length with less, sparse number of ganglions (*cone*); 3. A still more proximal, *hypertrophied dilated segment* is actually the normal ganglionic area.

Clinical features: It is common in males. In 90% of cases symptoms appear in early neonatal period, i.e. within three days of birth. Present with complaints of failure of passage of meconium. After introducing finger into the rectum child passes toothpaste like stool, with evidence of straining. Distension of the abdomen with features of intestinal obstruction is seen. Constipation with history of passing stools once in 3-4 days with straining, is seen throughout the childhood and also in adolescent period. Occasionally condition can cause intestinal obstruction.

Diagnosis: History of failure of passing meconium. Plain X-ray abdomen—shows intestinal obstruction. Biopsy from all three zones is taken to study the *ganglions and hypertrophic nerve terminals* in spasmodic segment. **Barium enema** is done to look for the extent of disease and three zones. Foley's catheter should not be used while doing barium enema in case of Hirschsprung's disease. **Anorectal manometry**—shows the absence of rectoanal reflex in Hirschsprung's disease, which is diagnostic.

Complications: Colitis; intestinal obstruction; growth retardation; constipation.

Differential diagnosis: Total neuronal dysplasia; Acquired megacolon; anorectal malformation.

Ulcerative Colitis

An inflammatory condition of rectum and colon of unknown aetiology perhaps related to stress, westernised diet, autoimmune factor, familial tendency, allergic factor. Disease commonly starts in the *rectum*, spreads proximally to the colon and often into the ileum as **back wash ileitis**.

Pathology: To begin with, multiple minute ulcers occurs, with proctitis and colitis → These ulcers extends into the deeper layer → Spasm of the bowel → Stricture of the colon → Permanently contracted

colon → In between ulcers, epithelial thickening occurs which appears like polyps → *Pseudopolyposis*.

Clinical features: More common in females, begins in 3rd decade. Watery diarrhoea, mucous or blood stained discharge per rectum; colicky pain, spasms; decreased appetite and loss of weight; relapses and remissions at regular intervals. Two types of presentations: (a) **Fulminant type**—5% common. It is a severe form, with continuous diarrhoea with passage of blood, mucous and pus. Patient is ill and dehydrated; mimics fulminant amoebic colitis, severe typhoid and dysentery. Later abdominal distension occurs. May go for *acute toxic dilatation* (1.5%) of *transverse colon* where in the diameter of transverse colon > 6 cm. It has high mortality and requires *emergency surgery*, i.e. either colostomy or resection with ileostomy and later ileoanal anastomosis. (b) **Chronic type**: (95%) Lasts for months and years with diarrhoea, blood loss, anaemia, invalidism, abdominal discomfort and pain.

Investigations: (a) *Barium enema*—shows loss of haustrations, narrow contracted colon (*hose pipe colon*), mucosal changes, pseudopolyps. It is avoided in fulminant cases. (b) *Sigmoidoscopy and biopsy*. Colonoscopy also is required. Due to very high incidence of malignant transformation in ulcerative colitis (10-20%), multiple biopsies should be taken from suspected areas of the colon. Risk increases with age of the patient and duration of the disease (20%).

Complications: Pseudopolyposis; turning into malignancy; stricture formation commonly in rectosigmoid and anal canal; toxic megacolon in transverse colon; massive haemorrhage; fistula in ano; liver cirrhosis (50%); skin lesions; arthritis; iritis, ankylosing spondylitis; sclerosing cholangitis, carcinoma of gallbladder.

Familial Adenomatous Polyp (FAP)

It is inherited as an autosomal dominant neoplastic condition (chromosome no.5). Incidence is equal in both sex, involving commonly the large intestine but can also occur in stomach, duodenum and small intestine. It is familial with a *high potential for malignant transformation*. It can be associated with duodenal

or ampullary carcinomas, *Gardner's syndrome* (Desmoid tumour in the abdomen, osteomas (75%) and epidermoid cysts) and also *Turcot's syndrome* (FAP + brain tumour (medulloblastoma or gliomas)). It presents in younger age group - 15-20 years; usually multiple (over 100); presents with lower abdominal pain, loose stools with blood and mucous, weight loss. If there is no adenoma at the age of 30 years, then it is not FAP of colon.

Carcinoma Colon

It is commonly *adenocarcinoma*. Very rarely adenosquamous, squamous carcinoma can occur.

Adenocarcinoma: Sigmoid colon (21%) is the commonest site of malignancy after rectum (38%). In caecum it is 12% common.

Aetiology: Diet: Red meat and saturated fat increases the incidence of colonic cancer. Cholesterol increases the bile acid concentration in the intestinal lumen which acts as cocarcinogen. High fibre diet protects the colon against cancer. **Genetic:** Carcinoma colon is more common in individuals with *adenoma colon* or with *familial adenomatous polyposis*—FAP or with *long standing ulcerative colitis*. Alcohol and cigarette smoking increases the risk. *Aspirin and other NSAIDs protect against colonic cancer*.

Types: Patient can have *de novo multiple primary* carcinomas in different parts of the colon at same time, i.e. **synchronous (5%)**, or can present with growth in different parts of the colon in different periods, i.e. **metachronous (2-5%)**. **Gross types:** Annular (stenosing)—It is more common on left side. Tubular; Ulcerative (common on right side); Cauliflower-like. Here the growth spreads round the internal wall and so it often presents with intestinal obstruction.

Spread: Locally it can invade the bladder, obstruct ureter and so cause hydronephrosis. It can perforate and cause peritonitis/ pericolic abscess/ faecal fistula. Growth may get adherent to psoas muscle posteriorly. Growth through lymphatics spreads to pericolic, epicolic, intermediate and principal group of lymph nodes; 40% of carcinoma colon spreads to *liver via portal veins*. *Secondaries* may be either *solitary or multiple*, presents as enlarged liver with hard, umbilicated nodules. Rarely it spreads to *bone, lung, skin*.

Clinical features: It occurs usually after 50 years. Familial type can present in younger age group. Commonly presents with loss of appetite and weight, anaemia, abdominal discomfort and mass per abdomen. 20% of cases presents as an acute intestinal obstruction. **Right sided growth** commonly presents with *anaemia, palpable mass* in the right iliac fossa, which is not moving with respiration, mobile, nontender, hard, well localised with impaired resonant note. Carcinoma caecum occasionally can present like acute appendicitis or intussusception with intestinal obstruction. **Left sided growth** presents with *colicky pain, altered bowel habits (alternating constipation and*

diarrhoea), palpable lump, and distension of abdomen due *subacute/chronic obstruction*. Later may present like complete colonic obstruction. Tenesmus, with passage of blood and mucous, with alternate constipation and diarrhoea, is common. Bladder symptoms may warn colovesical fistula. Enlarged liver with multiple umbilicated hard secondaries, ascites, rectovesical secondaries, palpable left supraclavicular lymph nodes are other presentations. Faecal strength of *Streptococcus bovis* bacteria increases many fold in colonic cancer patients compared to individuals without colonic cancer.

21

Examination of Mass Abdomen

In a patient presenting with abdominal mass, generally following history should be elicited carefully.

Pain: Site, nature, aggravating or relieving factors, duration of pain, referred pain.

Vomiting: Type, content, haematemesis, relation to food, frequency.

Jaundice: It is an important factor in relation to liver, gallbladder or pancreatic masses.

Bowel habits: Constipation, diarrhoea, bloody diarrhoea, furious diarrhoea, tenesmus.

Decreased appetite and weight.

Inspection of the mass: Anatomical location, margin, surface, movement with respiration.

Palpation of the mass: Site, extent, surface, tenderness, consistency, movement with respiration, mobility, borders, plane of the swelling (by leg rising test), presence of other masses.

Percussion: It is an important aspect of examination in case of an abdominal mass. Percussion over the mass is important to determine the anatomical location of the mass. If mass is dull, then it lies in the anterior abdominal wall or intra-abdominally in front of the bowel, liver, spleen, gallbladder, etc. If the mass is with an impaired resonant note, then the mass is arising from the bowel like stomach, colon, and small bowel. If the mass is resonant on percussion, then the mass is probably in the retroperitoneal region. Other than this, liver dullness, free fluid in the abdomen should be elicited during percussion.

Per-rectal examination: It is done to look for any secondaries in rectovesical pouch, any primary tumour or relation of lower abdomen masses (pelvic masses).

Pervaginal examination: It is done to assess pelvic masses.

Abdomen is divided into nine regions by four lines (Fig. 21.1).

Upper horizontal or *transpyloric line* is midway between the suprasternal notch and symphysis pubis or line between tips of ninth costal cartilages on each side. It is often midway between xiphisternum and umbilicus.

Lower horizontal line is *transtubercular line* at the level of two tubercles (5 cm behind the anterior superior iliac spine along the iliac crest) on the iliac crest.

Right vertical line is the line through the midpoint of right anterior superior iliac spine and pubic symphysis. It is usually a line joining right midclavicular and right midinguinal points.

Left vertical line is the line through the midpoint of left anterior superior iliac spine and pubic symphysis. It is usually a line joining left midclavicular and left midinguinal points.

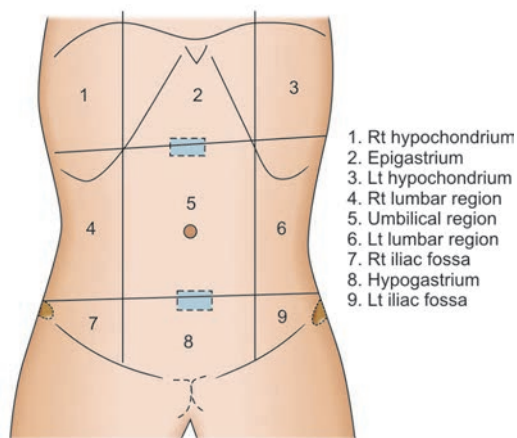


Fig. 21.1: Different regions in the abdomen.

Regions in the abdomen

Right hypochondrium
Epigastrium
Left hypochondrium
Right lumbar region
Umbilical region
Left lumbar region
Right iliac fossa
Hypogastrium
Left iliac fossa

Quadrants in the abdomen are four in number formed by two lines—one is vertical midline through the umbilicus; another is horizontal line passing through the umbilicus. Quadrants are—right upper, right lower, left upper and left lower (**Fig. 21.2**).

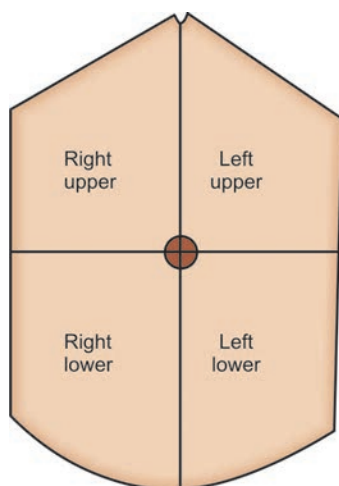


Fig. 21.2: Different quadrants in the abdomen. They are four in number formed by two lines—one is vertical midline through the umbilicus; another is horizontal line passing through the umbilicus. Quadrants are—right upper, right lower, left upper and left lower.

Chief Complaints

- Mass per abdomen—ask for duration, progress, site, mass appearing/disappearing (like in intussusception, Dietl's crisis of hydronephrosis kidney, and choledochal cyst)
- Pain in the abdomen—region of pain; duration of pain to be mentioned
- Vomiting—duration
- Haematemesis, malaena—duration
- Satiety—sensation of fullness after taking food (early satiety signifies gastrointestinal pathology like carcinomas)

- Yellowish discolouration of sclera—duration
- Loss of appetite and decreased weight—weight loss more than 10 Kg in short period/6 months is significant
- Altered bowel habits/constipation/diarrhoea
- Fever—Its character is important to be noted in abdominal tuberculosis, amoebic liver abscess, cholangitis, malignancy with tumour necrosis, infected pseudocyst of pancreas.

History

History of Present Illness

Pain

Site of origin of pain; onset (sudden/insidious); duration; radiation of pain/referred pain; type of pain—intermittent/persistent; dull, severe pricking, colicky; periodicity with an interval of free period—ulcer pain has often got periodicity unless it is complicated; relation to food intake—more/less/not related to meals; relation to vomiting/induced vomiting; aggravating/relieving factors; pain in relation to bowel habits/urinary habits.

Vomiting

- Duration, frequency, relation to food, type (projectile/effortless)
- Vomitus—content (food/blood/bile), quantity, smell, colour—coffee ground/bloody/yellow, taste
- Relation to pain, details of haematemesis if present
- It is better to ask the patient to collect and keep the vomitus and clinician should personally observe it.

Jaundice

- Duration, colour (greenish yellow suggests obstruction), severity, progress (progressive/intermittent/static/reducing)
- Presence of fever with jaundice—cholangitis
- Association with pruritus, clay coloured stool/silvery stool.

Altered Bowel Habits

- Duration, type, malaena, with distension of abdomen

Altered Urinary Symptoms

History of frequency/urgency/haematuria/pyuria/oliguria/painful urination/burning urine/difficulty in passing urine/hesitancy/hiccough/oedema feet or face; relation of urinary symptoms to pain, mass in abdomen should be asked for.

Other Relevant History

Cough and haemoptysis, bone pain, etc.—suggestive of metastases.

Past History

Earlier history of abdominal surgery—reason for surgery, how long ago it was done, whether earlier symptoms are relieved or not, whether the symptoms are now similar or different, whether it was an emergency or an elective surgery, whether it was earlier properly investigated or not, whether drain was placed or not—if placed when it was removed; what content was coming through the drain, whether blood transfusion was done during surgery or in postoperative period.

Personal History

History of alcohol intake, diet, smoking, etc. has to be noted.

Treatment history—any relevant history of surgery in the past, chemotherapy for malignancies, abdominal tuberculosis treated, and so on.

Family history—any relevant history in the family should be taken as some GI malignancies run in families.

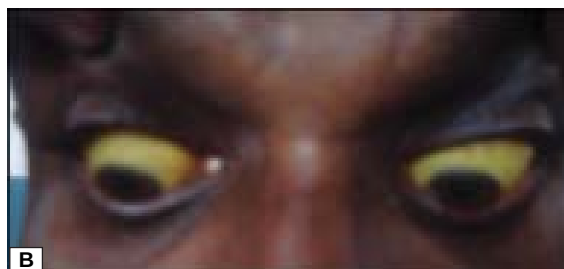
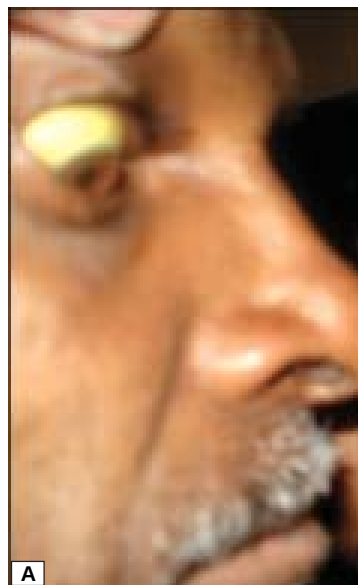
General Examination

Pallor/jaundice/clubbing/oedema feet /cachexia is noted. Pulse/blood pressure is recorded. Genitalia/respiratory and cardiovascular system should be examined (**Figs 21.3A and B**).

Local Abdominal Examination

Inspection

Inspection of the abdomen is done in supine position exposed from midchest to midhigh region with arms extended. Inspection is done from side of the bed as



Figs 21.3A and B: Obstructive jaundice in a patient with carcinoma head of pancreas. Note the sclera for discoloration. Severe itching is common in these patients.

well as from foot end with eye level at the level of the patient (**Figs 21.4A to 21.5B**).

Shape of the abdomen—contour—normal/scaphoid/distended.

Skin over the abdomen whether stretched/pigmented; presence of scar whether healed primarily or secondarily; site of scar; length and width of scar; whether there is any incisional hernia or not.

Dilated veins over the abdomen should be looked for—*caput medusae* is dilated veins radiating from the umbilicus—seen in portal hypertension. In *inferior vena caval obstruction* (lateral abdominal wall) dilated veins are visible with their flow of blood from below upwards towards superior vena cava. In *superior vena caval obstruction* dilated veins are visible with blood flow from above downwards. Dilated veins should be inspected in standing position and also



Figs 21.4A and B: Proper exposure of the abdomen is important from midchest to midhigh and position of the patient for proper abdominal examination.



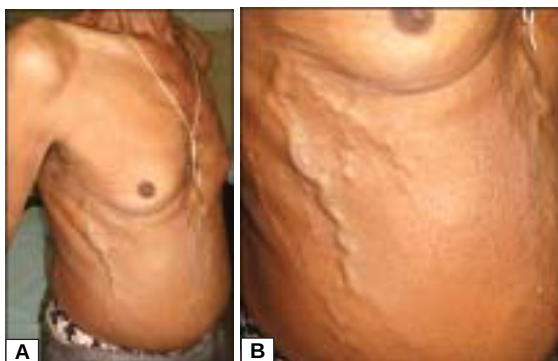
Figs 21.5A and B: Inspection of the abdomen should be done at the level of the patient's abdomen both from right side as well as from foot end.

direction of flow should be checked by placing two fingers apart over the vein and the fingers are released one by one to see the direction of blood flow. Normally abdominal wall drains to superior vena cava above the umbilicus and to inferior vena cava below the umbilicus—*water shed area* (**Figs 21.6 and 21.7A and B**).

Movements of regions with respiration should be noted.



Fig. 21.6: Superior vena caval obstruction causing dilated veins in the neck chest wall and shoulder. Note the neck swelling extending into the mediastinum.



Figs 21.7A and B: Inferior vena caval obstruction causing dilated veins over the lateral aspect of the flank with flow of blood upwards.

Pulsations over the mass or any region should be noted. Patient should hold the breath after full expiration to see for pulsations.

Any visible peristalsis should be looked for—*Visible gastric peristalsis (VGP)* is seen in upper middle region with waves beginning from left upper abdomen directed downwards and towards right to umbilical region. It is stimulated by drinking glass of water or by massaging the epigastrium. It signifies gastric outlet obstruction. But may be absent in gastric outlet obstruction where gastric paresis develops and stomach becomes dilated and silent without any motility. *Visible intestinal peristalsis (VIP)* occurs in *step ladder* pattern in central abdomen from left to right or vice versa

in umbilical region. Visible colonic peristalsis may be obvious from right to left along the line of colon

Inspection of the mass—Its location (exact location should be mentioned as in which region it is located and then its extension into the other region should be mentioned later); extent; approximate size; well defined or ill defined (often mass is not clearly seen but fullness is visible); margins whether clear or not or which part is clear and which part is not; presence of movement of mass with respiration or not (upper abdomen mass like liver, stomach, spleen, gallbladder, omental mass, kidney mass moves with respiration). Mass which was initially mobile may not be mobile later once it gets fixed to retroperitoneum or deeper plane. But occasionally mass which was initially not mobile, may start moving with respiration once gets attached to structures like omentum. Lower abdominal mass, retroperitoneal mass will not usually move with respiration. Mass which comes in close contact with diaphragm will move with respiration. Composite mass may move with respiration because of its component like omentum, lymph nodes, bowel, etc (**Figs 21.8 to 21.10**).

Umbilicus—Position is noted. It may be everted/inverted. *Tanyol sign*: Umbilicus is shifted upwards in pelvic/ovarian mass and downwards in ascites. *Sister Joseph nodules* can occur in the umbilicus as secondaries from abdominal GI malignancies through ligamentum teres. Umbilical black eye is *Cullen's sign* of discolouration of umbilicus seen in acute pancreatitis. Umbilical concretions, umbilical discharge



Fig. 21.8: Visible large upper abdomen mass—could be enlarged liver/pseudocyst of pancreas/retroperitoneal mass.



Fig. 21.9: Large secondaries in liver. Patient has undergone enucleation of left eye (with artificial eye) for primary melanoma choroids—15 years ago. Now he has presented with late large liver secondaries.



Fig. 21.10: Head raising test should be done to find out whether mass is intra-abdominal or in the abdominal wall.

(sinus/fistula), bluish tinge in ruptured ectopic gestation (*Cullen's*), yellow tinge around umbilicus in acute pancreatitis in women (*Johnston*)—should be observed (**Fig. 21.11**).

Hernial orifices and genitalia inspection—is a must. Scrotum should be examined for testicular tumour/



Fig. 21.11: Sister Joseph secondary nodule in the umbilicus.



Fig. 21.12: Impulse on coughing should be seen and also felt to confirm associated hernia.

loss of testicular sensation as testicular tumour may present as epigastric mass due to enlarged para-aortic lymph nodes (**Figs 21.12 and 21.13**).

Palpation

While palpating the abdomen patient should take deep breath with mouth open to relax the abdomen otherwise it is difficult to get proper finding. Hands should be warm and forearm should be horizontal at the same level as patient's abdomen. Palpation is done with ventral aspect of the fingers. Legs should be partially flexed at hips and knees.



Fig. 21.13: Loin should be inspected from behind for fullness and oedema.

Local rise of temperature is checked using back of hand. It suggests inflammatory pathology.

Tenderness over the abdomen or over the mass must be noted. It may be due to inflammatory pathology. Often malignant condition may cause tenderness either due to secondary infection or due to tumour necrosis.

Position, size, shape, and surface of the mass: Nodular surface may be neoplastic; smooth surface may be of benign or inflammatory pathology.

Margin: Well-defined margin which is distinct may be a feature of neoplasm. Ill-defined margin may be seen in inflammatory or traumatic pathology. Margin which is indistinct whether upper or lower should be confirmed. In the upper abdomen feeling the upper margin is important. In liver mass upper margin is not felt but it is felt in stomach mass. Upper margin of the mass may be difficult to feel in mass from fundus of stomach. Feeling the lower margin is important in the lower abdomen mass. If lower margin is not clear one has to find out whether mass is extending to pelvis or not. Rectal or per vaginal examination confirms the pelvic mass. Often full bladder may interfere or mimic the mass and so mass should be palpated again after emptying the bladder, if needed after passing

a urinary catheter. Margin may be better felt with change in position either sitting, standing, or lateral position.

Mobility of the mass: Mass is held between thumb and fingers and moved in vertical and horizontal directions. If there is restriction in movement, which movement is restricted should be checked. Totally fixed mass will not be mobile at all.

During inspiration (on deep breathing) mass moves down to touch the hand of the examiner kept on the lower margin of the mass. During expiration it moves back to its original position (**Figs 21.14 to 21.16**).



Fig. 21.14: Checking the temperature of the abdomen using dorsum of the hand.



Fig. 21.15: Palpation of abdominal mass using fingers.

Head raising test or leg raising test (Carnett's test):

It is done to confirm whether mass is in the abdominal wall or intra-abdominal. Mass is seen initially and palpated and patient is asked to raise his head along with shoulders with arms folded over the chest. If mass disappears or becomes smaller, it is intra-abdominal mass; if becomes more prominent it is in the abdominal wall. Manoeuvre is done to make the abdominal muscle



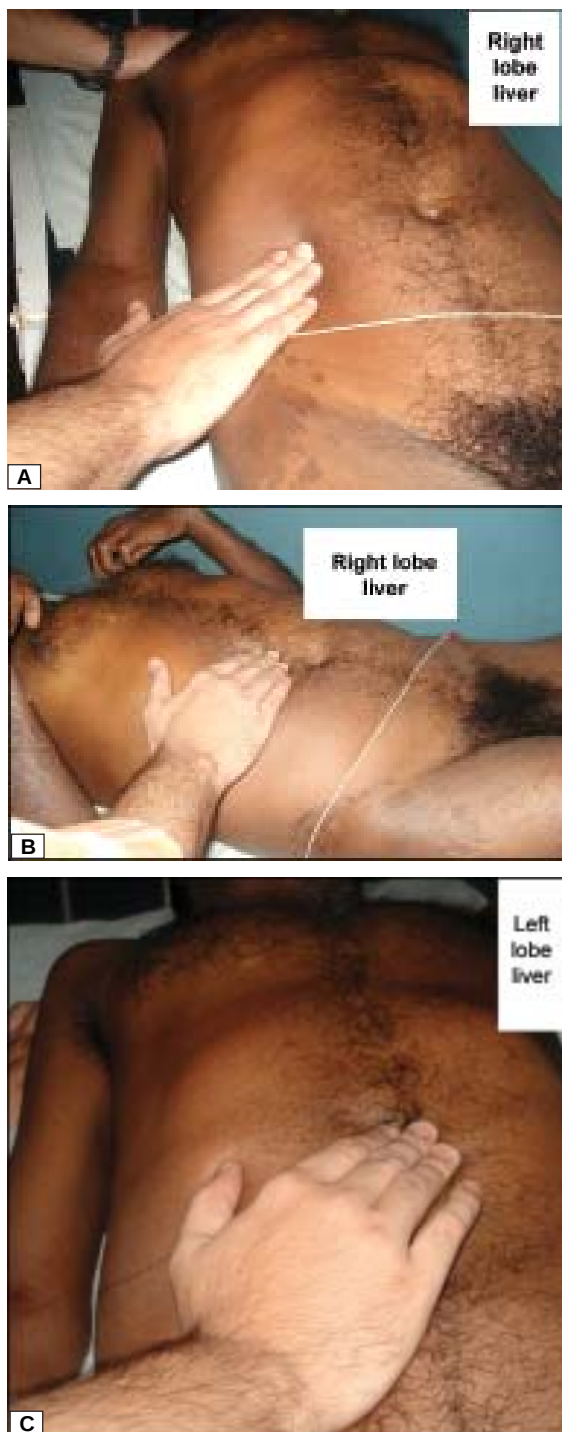
Fig. 21.16: Lower abdominal mass—retroperitoneal tumour/ovarian tumour/uterine mass.

taut. Raising both legs straight above the bed (Carnett's test) can also be used for the same. Air is tried to be blow out by holding the nose tightly with fingers and mouth shut—*Valsalva manoeuvre*. Abdominal wall mass will become prominent and immobile during these manoeuvre.

Palpation of Liver

Liver is palpated by placing flat of the hand parallel to the right costal margin—initially near right iliac fossa with fingers directed upwards up to the margin of the right rectus. Slowly with each phase of respiration fingers should be moved upwards towards right hypochondrium to feel the lower margin of the liver. The surface of the liver is then felt for tenderness, nodularity, round/sharp margin. Level of lower margin should be measured in centimeters from right costal margin. In children below 3 years, liver is palpable 3 cm below the right costal margin. Liver is not palpable or just palpable in normal adult. Whenever there is ascites liver is palpated by '*dip method*' - (dipping fingers quickly so as to displace the fluid). Liver may be enlarged *upwards* in hydatid cyst, and liver abscess (**Figs 21.17A to C**).

Normal liver span in adult is (vertical height) 12-15 cm. Liver span in infant is 2.4-2.8 cm. At the age of 14 years it is 5.5-7.5 cm.



Figs 21.17A to C: Method of palpating the liver right lobe and left lobe.

Palpation of Gallbladder

Normally it is not palpable. When enlarged its lower margin may be in right side of umbilical region/right lumbar region/right iliac fossa. It moves with respiration, globular in shape, smooth and soft, may be horizontally mobile but not vertically, upper margin merges under the liver when liver is enlarged or under the right costal margin. It is usually in right hypochondrium, just right of the right rectus muscle.

Murphy's sign is elicited in sitting position. Patient winces with pain at the summit of inspiration while palpating in gallbladder area. During deep inspiration, inflamed gallbladder comes down and touches the palpating finger (thumb) to cause tenderness. It is observed in chronic cholecystitis. Often it is elicited in lying down position and is called as *Moynihán's sign* (test).

Stomach

Stomach is palpated in the epigastrium. Entire stomach may be dilated and palpable due to gastric outlet obstruction. Succussion splash and auscultopercussion tests should be elicited in such occasion.

Succussion splash: Patient should not take anything orally for 4 hours as gastric emptying time for liquid is 4 hours. If patient drinks fluid succussion splash may be positive even when stomach is not dilated. Bell of the stethoscope is placed in the epigastrium. Two thumbs of the two hands are placed over the bell and fingers of each hand are placed on costal area on each side and shaken well to hear the splashing sound. This can be occasionally elicited by dipping the hand over the dilated stomach also (**Fig. 21.18**).

Auscultopercussion test: It is positive in gastric outlet obstruction. Bell of the stethoscope is placed over the epigastrium. Abdominal wall is scratched using pencil or fingertip by radiating strokes from bell area towards left hypochondrium, left lumbar and left and later towards right part of the umbilical regions. Change in the note of the sound is marked at each stroke line. All these marks are joined to mark the greater curvature of the stomach. By this procedure only greater curvature is assessed. Reasons are—only greater curvature dilates significantly when there is obstruction not lesser curvature, and greater curvature



Fig. 21.18: Succussion splash

is more towards surface whereas lesser curvature is in deeper plane. Normally greater curvature is above the level of umbilicus on surface marking. In gastric outlet obstruction it shifts below the level of umbilicus (**Figs 21.19A to C**).

Stomach mass is commonly due to carcinoma stomach but occasionally it can be due to gastric lymphoma or leiomyoma of stomach. Mass of carcinoma stomach is in the epigastrium or upper part of umbilical region—which moves with respiration; all borders are well made out; mobile in all directions; nodular and hard; upper border is well made out; with impaired resonant on percussion. If mass is close to the fundus of stomach then upper border may not be clearly felt. Often patient should be examined in lateral position or after making the patient to walk for few minutes so as to allow the mass to come down to make it easily palpable. When mass arises from the pylorus it will be just above right of the umbilicus presenting with features of gastric outlet obstruction. Mass from the body of the stomach is horizontally placed extending towards the left hypochondrium, commonly without features of obstruction. Often a composite mass of carcinoma, lymph nodes, omentum and part of the liver may be



Figs 21.19A to C: Auscultopercussion test.

palpable and attains a large size also. Carcinoma stomach when fixed may not move with respiration and may find it difficult to differentiate from pancreatic mass even though carcinoma pancreas is rarely palpable. In case of palpable gallbladder and progressive severe jaundice one should suspect carcinoma pancreas. Often carcinoma stomach can also cause jaundice when there are secondaries in liver extensively in both lobes. In such occasion along with stomach mass nodular secondaries in liver with ascites is also evident. Patient with mass near the oesophago-gastric junction presents with dysphagia. *Linitis plastica* (diffuse type of carcinoma stomach in submucosal plane) usually presents as loss of appetite and decreased weight with reduced stomach capacity. It usually does not present as mass abdomen. When mass is palpably present, it is a composite mass of nodes, omentum and stomach. It carries poor prognosis. Total gastrectomy is the treatment. Clinically palpable carcinoma stomach (as mass) is considered as advanced carcinoma stomach as involvement of serosa means 'advanced' as per the definition. Without serosal breach it is difficult to palpate clinically. But it could be surgically resectable (**Figs 21.20 and 21.21A to E**).

In infants pyloric mass of congenital pyloric stenosis is palpated from *left side* of the patient.

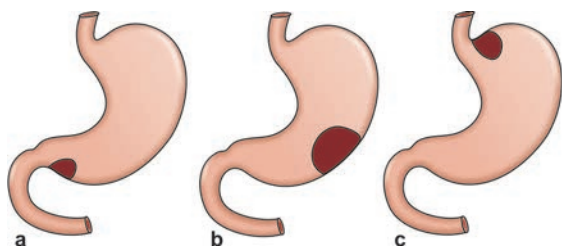


Fig. 21.20: Different locations of carcinoma stomach. (a) Pylorus, (b) Body of stomach, (c) Near OG junction.

Pancreatic mass

It is palpable in the epigastrium. It is deep, nonmobile, not moving with respiration, with bowel in front. It is felt on deep palpation. Pseudocyst mass has rounded lower margin with transmitted pulsation. Pancreatic masses are usually resonant.

Palpation of Spleen

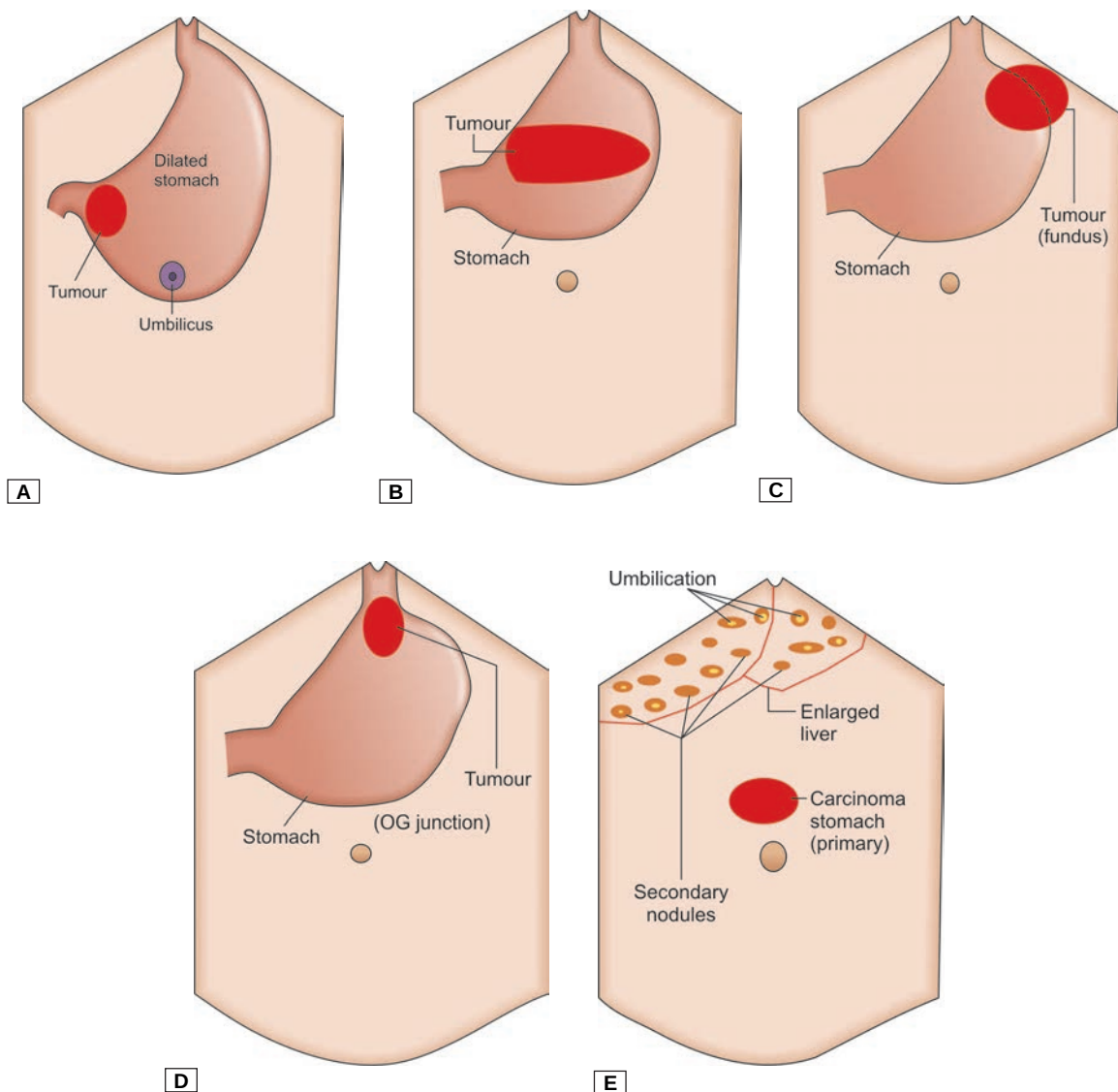
Spleen is normally not palpable. It becomes clinically palpable when it is enlarged more than 2½ times. Non-palpable spleen still could be enlarged. Spleen enlarges towards right iliac fossa across umbilical region directing obliquely—downwards, forwards, inwards. It is palpated by placing fingers of right hand over right iliac fossa with left hand under left costal margin for support. Fingers of right hand are gradually and gently moved towards left hypochondrium during phases of respiration to feel the splenic upper margin near anterior end often with a *notch* (notch need not be present always). Fingers cannot be insinuated under the left costal margin. Spleen is smooth, firm in consistency, moves with respiration, and usually non tender unless massively enlarged. Often patient need to be tilted towards right side to palpate the spleen easily. It can be palpated from left side by hooking the left costal margin—*hook sign* (also refer pg 466, Chapter 20). (Spleen is dull on percussion) (**Figs 21.22A to E**).

Palpation of Kidney

Loin mass: If loin mass is present, it should be checked by *bimanual palpation*. Patient in lying down position, right side is examined from right side; left side from left side. Right hand is placed over the abdomen in front of lumbar region; left hand is placed behind the region. Fingers of both front and back hands are approximated to compress the loin. Mass if present is felt properly. Its size, shape, tenderness, surface, consistency, movement with respiration, mobility, medial extent should be checked.

Ballottability: It is again done for loin mass. Right hand fingers are placed in front; left hand fingers are placed behind. Left hand fingers are pushed forward from behind so that loin mass is pushed forward. Examiner can appreciate that mass is moving forward and touching his fingers in front. Ballottability is due to soft perinephric pad of fat and pedicle on the medial side on which kidney moves/rotates. Kidney mass is bimanually palpable and ballottable. If there is perinephric inflammation causing adhesions or renal cell carcinoma infiltrating the perinephric tissues kidney will be only bimanually palpable not ballottable.

When kidney is enlarged, it will be *bimanually palpable, ballottable* (left hand from behind is pushed anteriorly and kidney can be felt moving forward and



Figs 21.21A to E: Carcinoma pylorus causes gastric outlet obstruction with palpable mass above the umbilicus. Carcinoma body of stomach mainly presents as loss of appetite and decreased weight with horizontally placed stomach mass. Carcinoma from fundus of the stomach presents as mass abdomen with loss of appetite and weight. Carcinoma OG junction presents as dysphagia. Carcinoma stomach is one of the common causes of secondaries in liver.

touching/pushing the right hand in front), moves with respiration (as it is related to diaphragm), vertically placed with resonant colonic band in front because of medial and anterior push of the colon by enlarged kidney. It is smooth and soft in hydronephrosis; hard and nodular in carcinoma kidney; firm, nodular and

bilateral in polycystic kidney disease. Kidney may not move or may not be ballottable if it is adherent due to infection or advanced carcinoma. Hand can be insinuated between upper part of the mass and right costal margin. It usually does not cross the midline (to opposite side).



Figs 21.22A to E: Method of palpating spleen and also eliciting hook sign.



A



B



C

Figs 21.23A to C: Renal angle should be palpated and percussed in a kidney mass—in sitting position.

Murphy's kidney punch is eliciting the tenderness in renal angle in *sitting position* from behind. In sitting position from behind loin should be inspected for any fullness. *Renal angle* tenderness is elicited using thumb pressing at the angle (renal angle is between erector spinae muscle and 12th rib). Renal angle also should be percussed to look for any change in note. Normally it is resonant because of the ascending/descending colon which is displaced by enlarged kidney making it dull on percussion (**Figs 21.23A to 21.25**).

Note

Renal mass loses all its features once it is fixed and advanced. Intraperitoneal mass once adherent posteriorly to retroperitoneum behaves clinically like a retroperitoneal mass.



Fig. 21.24: Palpation for kidney mass—for ballottability and bimanual palpation.



Fig. 21.25: Renal bruit should be auscultated.

Small bowel mass

It is felt as mobile, localised mass with resonant or impaired resonant note. It does not move with respiration. Intussusception is sausage shaped mass with concavity towards umbilicus. It appears and disappears; contracts under the palpating finger (**Fig. 21.26**).



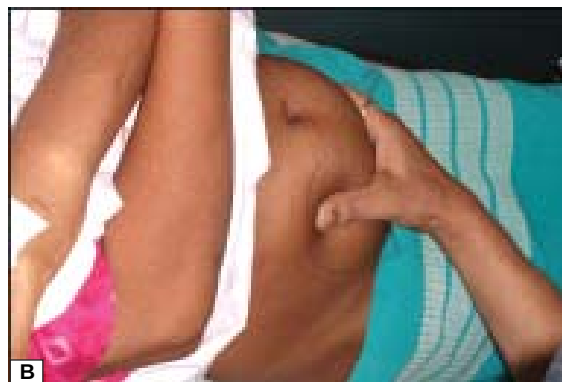
Fig. 21.26: Often abdominal mass should also be examined in lateral position to get better feeling and findings.

All masses in the lower quadrants should be palpated *after emptying bladder* or passing a urinary catheter. Upper border is clearly felt but not lower border which merges into the pelvis. Mass also should be *bimanually palpable* by placing fingers in rectum or per vagina (**Figs 21.27 to 21.29**).

External genitalia should be palpated for any swelling/loss of testicular sensation, secondary hydrocele.



Fig. 21.27: Palpating the lower border of the mass is very important in lower abdominal masses. Bladder should be emptied or catheterised before palpation.



Figs 21.28A and B: Intrinsic mobility of the mass should be checked in all abdominal masses.



Fig. 21.29: External genitalia should be palpated for mass/hydrocele.

Often there may be more than one mass in the abdomen. So when one mass is felt always look for *other relevant masses*.

Retroperitoneal masses and pulsatile mass like aneurysms should be examined in knee-elbow/knee—chest position. Retroperitoneal mass will not fall forward whereas intra-abdominal mass will fall forward. *Aortic aneurysm* with expansile pulsation will retain its pulsation whereas mass with *transmitted pulsation* will show reduced/absent pulsation in knee elbow position (**Figs 21.30A to C**).



Figs 21.30A to C: Knee elbow position—palpation of retroperitoneal mass. Mass can be held to check mobility, relations.

Percussion

Liver dullness should be assessed by percussion. It is done by percussing from above downwards over right intercostal spaces in midclavicular line. Liver span also can be assessed by this (**Fig. 21.31**).



Fig. 21.31: Liver dullness should be assessed (upper border of liver) by percussing from above downwards in intercostal spaces in midclavicular line and space is marked.

Percussion over the mass is very important. Mass in front of the bowel is dull on percussion like parietal/abdominal wall mass, liver, spleen, gallbladder, etc. Mass from the stomach/small bowel/colon shows impaired resonance on percussion. Mass from retroperitoneum shows resonance on percussion.

Hydatid thrill is elicited using three fingers. Index, middle and ring fingers are placed over the liver mass with gaps between each fingers. Percussion is done over the middle finger to feel the fluid thrill over other two fingers (**Fig. 21.32**).



Fig. 21.32: Percussion over the mass is essential to say whether mass is anterior to bowel (dull); from the bowel (impaired resonant) or behind the bowel (resonant)

Percussion for free fluid is important. With patient in supine position, percussion is done over the epigastrium initially to confirm resonant note. Then percussion is continued over to one side flank until one gets dullness. Patient is tilted towards opposite side to make area of percussion directed upwards so as to displace the fluid from that side. After 1-2 minutes (time to allow fluid to shift towards opposite side) without removing the fingers same area is percussed to get resonant note which confirms the presence of fluid. For massive ascites, fluid is confirmed by eliciting fluid thrill. Patient is asked to place his hand over the epigastrium with ulnar side of the hand pressed firmly in midline. Examiner should keep his one hand over one lumbar region of the patient and with fingers of other hand, opposite lumbar region is tapped to elicit fluid movement as *fluid thrill*. Small quantity of fluid can be detected in knee elbow position. In this position umbilical site is percussed to elicit dullness which signifies positive *puddle sign*—*signifying minimal ascites* (Figs 21.33 to 21.35D).

Percussion over renal angle is done to look for resonance (normal) or dullness (abnormal). Normal renal angle is occupied by ascending (right) or descending (left) colon and is resonant. When kidney is enlarged, it pushes the colon in front and medially replacing colon and so becomes dull on percussion. Renal angle is examined in sitting position between 12th rib and erector spinae muscle (Renal angle should be examined for fullness/tenderness/percussion note).



Fig. 21.33: Looking for minimal ascites in knee-elbow position—Puddle sign.



Figs 21.34A to C: Massive ascites. Eliciting fluid thrill in massive ascites.



Figs 21.35A to D: Confirming ascites/free fluid in the peritoneal cavity by percussion—classical method.

Auscultation

It is done for bowel sounds, bruit over the renal artery just to the side of the umbilicus, over the mass like liver which signifies vascularity, over aneurysm for bruit.

Left supraclavicular fossa between two heads of sternomastoid muscle should be palpated for Virchow's node enlargement—*Troisier's sign*—as secondary deposits.

Examination of respiratory system for effusion, altered breath sounds suggestive of metastases.

Examination of skeletal system - sternum, spine, skull and other bones for tenderness, swelling, pathological fracture, neurological deficits (**Figs 21.36A and B**).



Figs 21.36A and B: Spine and other skeletal system should be examined in patient with mass abdomen.

Digital Examination of Rectum (Per Rectal Examination/P/R)

Per rectal examination must be done in all cases of abdominal mass. It is done with patient in left lateral position with right leg flexed completely and left leg kept straight. Procedure is done after informing patient about the technique and taking consent. Xylocaine jelly is applied over the anus. It is inspected for discharge, opening, skin changes and swelling. Pulp of the gloved right index finger is gently pushed into the anorectum in the direction of the umbilicus. Sphincter tone is assessed. Posteriorly sacral curvature, rectal mucosa are assessed. Finger is turned towards front. Prostate, its texture, size, median groove are felt. Rectum is palpated for any growth, stricture or

MASS IN THE RIGHT HYPOCHONDRIMUM**Parietal swellings**

Sebaceous cyst, lipoma, neurofibroma, cold abscess (from ribs or spine, presents as soft, fluctuant nontender well localised swelling), liver abscess or subphrenic abscess rupturing into the abdominal wall presenting as parietal wall abscess

Intra-abdominal swellings

Liver: Congenital Riedel's lobe; amoebic hepatitis or liver abscess, portal pyaemia or pyogenic liver abscess, gumma of liver (large smooth hard hepatomegaly), hydatid cyst of the liver, hepatocellular carcinoma (HCC), secondaries in liver, early cirrhosis of liver or macronodular type.

Gallbladder: Mucocele, empyema, carcinoma, due to malignant CBD obstruction.

Subphrenic abscess

Pylorus of the stomach and duodenum

Hepatic flexure of the colon: Carcinoma; inflammatory mass

Right kidney: RCC, hydronephrosis

Right adrenal gland

secondary nodule in front above (as a hard nodule with free rectal mucosa—*Blumer shelf*). Gently finger is removed and fingertip should be inspected for content staining—blood/mucous/pus, etc. (**Figs 21.37A to C**). P/R is contraindicated in acute fissure in ano.

Per Vaginal Examination

It is done whenever pelvic mass is suspected with lower margin of the mass merging into the pelvis. Bimanual palpation is often done under general anaesthesia in lower abdominal masses.

Differential Diagnosis

Mass in any quadrant should be assessed first whether it is in the abdominal wall or intra-abdominal or retroperitoneal. From which specific organ mass is arising should be ascertained. Pathological nature of the mass must be assessed. So initially *anatomical diagnosis* is made and later *pathological diagnosis* is thought of, finally the *final diagnosis* is concluded.

Palpable liver Mass as Mass in Right Hypochondrium

It is horizontally placed; usually moves with respiration; upper border is not felt; it is dull on percussion; (this dullness continuous over liver dullness above); fingers cannot be insinuated under right costal margin.

Conditions where liver gets enlarged:

1. *Soft, smooth, nontender liver*—

a. **Hydrohepatosis:** It is due to obstruction of CBD causing dilatation of intrahepatic biliary radicles (usually malignant CBD obstruction but can occur in obstruction due to stones).



Figs 21.37A to C: Digital examination of the rectum is important (P/R; Per Rectal examination)

- b. Congestive cardiac failure.
 - c. Hydatid cyst of the liver—Here mass is well localised in the liver with typical hydatid thrill (Three fingers are placed over the mass widely. When central finger is tapped fluid movement elicited is felt in lateral two fingers) (**Figs 21.38A and B**).
 - d. Congenital Riedel's lobe is a tongue shaped projection from the lower border of the right lobe of the liver. It is often mistaken for enlarged gallbladder but is wider, flat and not spherical.
2. *Soft, smooth, tender liver*—
- a. Amoebic liver abscess: Here liver often gets adherent to the anterior abdominal wall and will not move with respiration. Intercostal tenderness



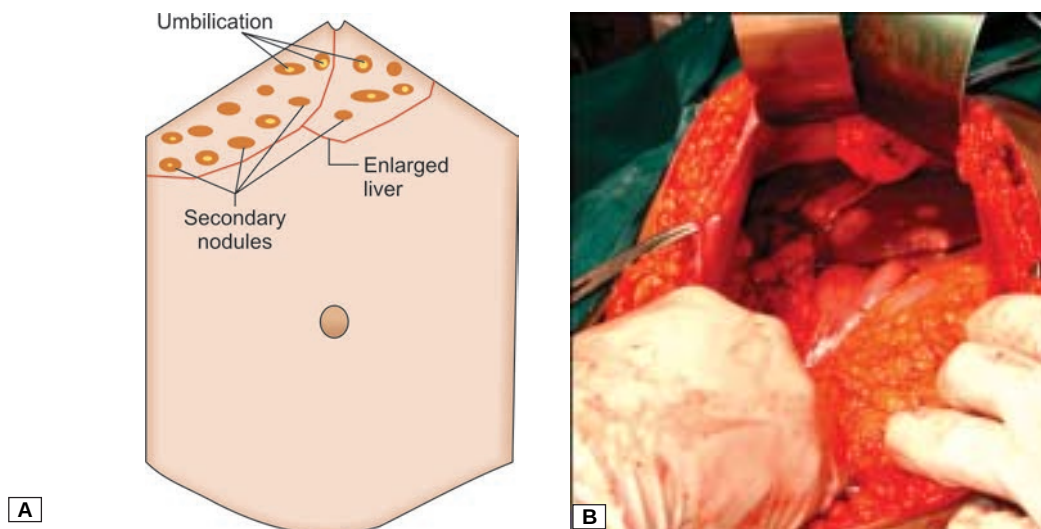
Figs 21.38A and B: Hydatid cyst of liver—fullness in right hypochondrium. Operated specimen of hydatid in same patient.

- and right sided pleural effusion is common. History of amoebic dysentery few months before may or may not be there. Fevers, referred pain in right shoulder, pallor, mild jaundice, elevation of upper border of the liver are the features. Subcutaneous pitting oedema in right hypochondrium is often very significant. X-ray will reveal the elevated diaphragm with pleural effusion. Amoebic hepatitis: Liver will be tender, smooth, soft or firm.
 - b. Viral hepatitis also causes smooth, soft, tender liver. Patient develops multiple joint pains in viral hepatitis.
 - c. Portal pyaemia: It causes tender soft liver with toxæmia, jaundice.
3. *Hard, smooth liver.*
- a. Hepatoma (IHCC): Here a large, single, hard nodule is palpable in the liver. But occasionally there can be multiple nodules when it is multicentric. Rapidly growing tumour can also be soft. Hepatoma often can also be tender due to tumour necrosis or stretching of the liver capsule. Vascular bruit may be heard over the liver during auscultation. It mimics amoebic liver abscess in every respect.
 - b. Solitary secondary in liver: It is not common but can occur (when primary is in colon); features of primary tumour may be present.
4. *Hard liver with multiple nodules.*
- a. Multiple secondaries in liver: Hard nodules here have umbilication which is due to central necrosis.
 - b. Macronodular cirrhotic liver or early cirrhosis.

Causes for massive liver enlargement: Gummatous liver (*Hepar lobatum*); secondaries in liver from melanoma; often large hepatoma. Melanoma especially primary from choroids can cause secondaries in liver as late as 15 years after therapy (surgery) for primary (**Figs 21.39A and B**).

Palpable Gallbladder in Right Hypochondrium

It is smooth and soft (except in carcinoma gallbladder); mobile horizontally (side-to-side); moves with respiration; located in lateral margin of the right rectus muscle, below the right costal margin or below the lower margin of the palpable liver; dull on percussion.



Figs 21.39A and B: Multiple secondaries in liver with umbilication. It is due to central necrosis. Secondaries are the commonest malignant tumour of the liver. It could be from GIT or extra gastrointestinal like from breast, lungs, melanoma, thyroid, prostate, kidneys, etc. Patient with liver secondaries have poor general condition. It should be differentiated from multicentric hepatoma. It is usually treated by palliative chemotherapy. Solitary secondary from carcinoma colon can be removed by segmentectomy. It has got poor prognosis.

Conditions where gallbladder is palpable:

1. *Soft, nontender gallbladder:*
 - a. Mucocele of the gallbladder.
 - b. Enlarged gallbladder in obstructive jaundice due to carcinoma head of the pancreas or periampullary carcinoma or growth in the CBD.
2. *Hard gallbladder:* Carcinoma gallbladder.
3. Smooth, tender, soft or firm gallbladder mass: Empyema gallbladder, acute cholecystitis mass.

Other Masses in the Right Hypochondrium

- a. *Pericholecystic inflammatory mass:* It is tender, smooth, firm or soft, not mobile, intra-abdominal mass often with guarding.
- b. *Mass arising from upper pole of the kidney:* It may be due to renal cell carcinoma or hydronephrosis.
- c. *Adrenal tumour* may be pheochromocytoma or adrenocortical carcinoma. It is nonmobile; does not move with respiration; extends medially; often crosses midline; fluctuating hypertension is common. Renal angle is normal and resonant. In children it could be neuroblastoma. Such neuroblastoma may cause secondaries in skull.

Note: Commonest benign tumour of liver is haemangioma.

Hepatoma/hepatocellular carcinoma/HCC

- Common aetiologies are aflatoxins, hepatitis B and hepatitis C virus infection, alcoholic cirrhosis, haemochromatosis, smoking, hepatic adenoma, clonorchis sinensis, polyvinyl chloride
- Unicentric and right lobe involvement is more common (**Fig. 21.40**)
- Fibrolamellar variant is common in left lobe, not related to hepatitis or cirrhosis without AFP level raise. There is increased serum vitamin B₁₂ binding capacity and neurotensin levels.
- It can be multifocal/indeterminate/spreading/expanding—*Okuda classification*
- Presents as large smooth hard liver mass—later jaundice, fever, pain and tenderness, ascites and bruit over mass
- Spreads by lymphatics, blood and direct spread
- Mimics amoebic liver abscess, secondaries, hydatid cyst, polycystic liver disease
- LFT, CT scan, raised AFP, liver biopsy are the investigations
- Hemihepatectomy in early operable growth is the treatment
- Hepatic artery ligation/intra-arterial chemotherapy/chemoembolisation/percutaneous ethanol or acetic acid injection/radiofrequency ablation/chemotherapy using adriamycin, carboplatin, gemcitabine—are palliative procedures.

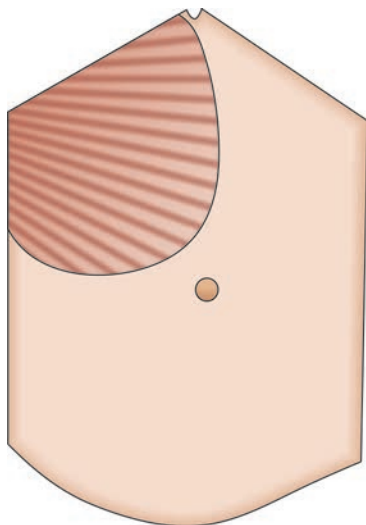


Fig. 21.40: Hepatocellular carcinoma/hepatoma. It is common in right lobe, unicentric, attains large size.

Amoebic liver abscess

- It is due to *Entamoeba histolytica* infestation
- It is more common in alcoholics and cirrhotics
- Single abscess is common—70%; common in right posterosuperior lobe—80% (**Figs 21.41 and 21.42**)
- Chocolate coloured *anchovy sauce* pus is classical
- Secondary infection can occur—30%—life threatening due to septicaemia
- It can be acute or chronic; both mimic hepatoma
- Rupture into lungs—commonest site of rupture
- Most dangerous rupture is into pericardium—left lobe abscess
- Liver failure can develop in cirrhotic patient
- Common in males (20:1), fever, pain, intercostal tenderness, tender liver—features
- Mimics cholecystitis, subphrenic abscess, hepatoma
- Total count, LFT, prothrombin time, US abdomen are relevant investigations
- Chest X-ray may show right sided sympathetic pleural effusion
- CT scan to differentiate from hepatoma
- Treatment—drugs like metronidazole, injection dihydroemetine, chloroquine tablets, diloxanide furoate; after controlling prothrombin time using inj vitamin K or FFP US guided aspiration; if recurs percutaneous guided drainage using pigtail catheter; open laparotomy and drainage with placement of Malecot's catheter



Fig. 21.41: Anchovy sauce pus in amoebic liver abscess.

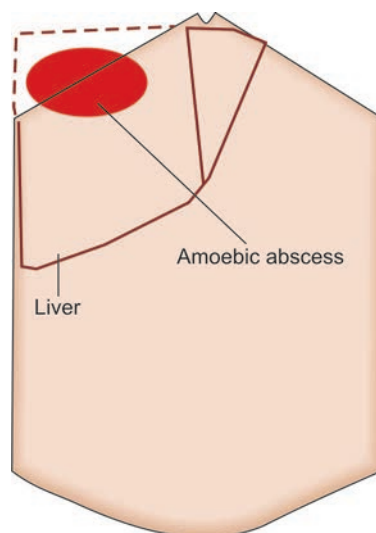


Fig. 21.42: Common location of amoebic liver abscess—posterosuperior part of right lobe.

Subphrenic Abscess

Subphrenic abscess presenting as mass is not common but mimics mass in the right hypochondrium. Common causes are postoperative sepsis, perforated duodenal ulcer, trauma.

Features: Pain, fever with often rigors, anorexia, nausea, tachycardia, tachypnoea. Pain may be present or may not be clear. When present it may be in the right hypochondrium/epigastrium/right thorax/lumbar region or may be referred to right shoulder. Tenderness in 11th intercostal space is very evident; tenderness

in right hypochondrium or in the loin is seen. Right sided pleural effusion can occur. Reduced breath sounds on right lower areas may be a feature. Jaundice is usually absent. There are 4 intraperitoneal and 3 extraperitoneal spaces. **Bernard** said—‘Pus somewhere, pus nowhere, pus under diaphragm’.

Courvoisier's Law

‘In a patient with jaundice if there is palpable gallbladder, it is not due to stones’.

In obstruction due to CBD stone, gallbladder does not distend because it is chronically inflamed, thickened and fibrotic.

In malignancy, like carcinoma of head of the pancreas or periampullary carcinoma, gallbladder will be distended and palpable to the right of rectus muscle in the right hypochondrium, as nontender, globular, smooth, soft, dull mass which moves with respiration and with horizontal mobility. *Exceptions for the rule are*—Absence of gallbladder; intrahepatic gallbladder; previous cholecystectomy; double impacted stone; large stone in Hartman's pouch.

Surgical Jaundice

Causes: Biliary atresia; choledochal cyst; CBD stones; ascending cholangitis; biliary strictures; sclerosing cholangitis; carcinoma of head and periampullary region of the pancreas; cholangiocarcinoma; *Klat skin tumour* (Carcinoma at the confluence of hepatic ducts above the level of the cystic duct and so will cause hydrohepatosis without GB enlargement); extrinsic compression of CBD by lymph nodes or tumours; parasitic infestations.

Classification of Causes of Obstructive Jaundice

Congenital: Biliary atresia, choledochal cyst

Inflammatory: Ascending cholangitis, sclerosing cholangitis.

Obstructive: CBD stones.

Neoplastic: Carcinoma of head or periampullary region of pancreas, cholangiocarcinomas, Klat skin tumour.

Extrinsic compression of CBD by lymph nodes or tumours.

Investigations for Obstructive Jaundice

Serum bilirubin. Normal value is less than 1.0 mg%. Both direct and indirect bilirubin is assessed. Direct is increased in obstructive jaundice, i.e. conjugated hyperbilirubinaemia. Vandenberg test is done.

Serum albumin, globulin and A: G ratio. Normal S. albumin is more than 3.5 gm%.

Prothrombin time. Normal value is 12-16 seconds. It is significant if it is more than 4 sec from the control or more than one and half times the control. It is corrected by injection vitamin K 10 mg IM od for 5 days or by fresh frozen plasma.

Serum alkaline phosphatase, SGPT, SGOT, 5' nucleotidase.

US abdomen.

ERCP to visualise site of obstruction, brush biopsy, bile sample for analysis.

MRCP—Noninvasive diagnostic tool.

CT scan in case of tumours to assess operability.

Urine tests: Fouchet's test for bile pigments, Hay's test for bile salts and test for urobilinogen in urine.

Fouchet's test: 10 ml of urine + 5 ml of BaCl₂ + pinch of MgSO₄ causes formation of BaSO₄ which is filtered over a filter paper and few drops of Fouchet's reagent is added. Green or blue colour signifies bile pigments in the urine.

Hay's test for bile salt: Sprinkle sulphur to 2 ml of urine. In presence of bile salts sulphur sinks to the bottom.

Ehrlich's test: 5 ml of freshly voided urine + 1 ml of Ehrlich reagent (p-dimethyl amino benzaldehyde) and wait for 5 minutes. Formation of red colour signifies presence of urobilinogen in urine. Normally it is present in traces, in obstructive jaundice it is absent and in haemolytic jaundice it is in excess.

Preoperative Preparation of Patient with Obstructive Jaundice

Proper diagnosis and assessment; Injection Vitamin K IM 10 mg for 5 days; Fresh frozen plasma—often requires 6 bottles or more; blood transfusion in case of anaemia; oral neomycin, lactulose; Mannitol 100-200 ml BD IV to prevent hepatorenal syndrome;

repeated monitoring by doing prothrombin time, electrolytes; antibiotics like third generation cephalosporins; Calcium supplements as calcium chloride IV.

Treatment of Obstructive Jaundice

CBD stones—ERCP stone removal, Choledocholithotomy, Transduodenal sphincteroplasty, Choledochojunostomy or Choledochoduodenostomy.

Carcinoma periampullary or head of pancreas—Whipples operation or Triple bypass or ERCPstenting.

Biliary stricture—Stenting, Choledochojunostomy, Roux en Y hepaticojunostomy.

Klat skin tumour—Radical resection or palliative stenting.

Biliary atresia—Kasai's operation or liver transplantation.

Choledochal cyst—Excision, hepaticojunostomy, mucosal resection.

Postoperative Management

Monitoring with prothrombin time, bilirubin, albumin, creatinine, electrolyte estimation; FFP or blood transfusion; Antibiotics; Observation for septicaemia, haemorrhage, pneumonia, pleural effusion, bile leak; Care of T tube and drains; T tube cholangiogram in 10-14 days; TPN, CVP line, nasogastric tube, urinary catheter.

Portal Hypertension

Sustained raise of portal pressure more than 12 mmHg. Isolated splenic vein thrombosis causes left sided sinistral/segmental portal hypertension.

Causes are—(a) Prehepatic—portal/splenic vein thrombosis, trauma, periportal inflammation, hypercoagulable status, neonatal umbilical sepsis. (b) Hepatic (80%)—cirrhosis, idiopathic, primary biliary cirrhosis, hepatitis, schistosomiasis, Wilson's disease, haemochromatosis, congenital hepatic fibrosis. (c) Post-hepatic—Budd-Chiari syndrome, constrictive pericarditis, veno-occlusive disease, congestive cardiac failure.

Presentations—Oesophageal varices (haematemesis/melaena), splenomegaly, ascites, jaundice, features of encephalopathy.

Investigations—Gastroscopy, LFT, splenoportography, US abdomen, CT abdomen, prothrombin time, liver biopsy.

Acute bleed is managed by pharmacotherapy (vasopressin, glypressin, octreotide, propranolol, Sengstaken-Blakemore balloon tamponade, surgical ligation of varices by various approaches.

Further bleeding is prevented by endoscopic banding for oesophageal varices; sclerotherapy; endoscopic glueing for gastric varices.

Shunt surgery is done if it is Child's grade A or B. Selective shunts like distal splenorenal shunt (Warren's shunt) or Inakuchi shunt between left gastric vein and IVC. Portocaval, mesentericocaval, proximal splenorenal shunts are nonselective shunts.

Orthotopic liver transplant is ideal and best. If patient is ideal for liver transplant open shunt surgery is contraindicated as liver hilum should be kept virgin for effective transplantation. *TIPSS*—Transjugular Intrahepatic porta Systemic Stenting can be done in these patients. TIPSS is a nonselective shunt.

Indications for Shunt Surgery: Child's Grades A and B

(Child's grading is used for selecting patients for surgery and predicting prognosis)

Surgery is contraindicated in Child C.

	Child A	B	C
Bilirubin	<2.0 mg	2.0-3.0 mg	>3.0 mg
Albumin	>3.5	3.0-3.5	<3.0
Ascites	None	Controlled	Uncontrollable
Mental status	Normal	Disoriented	Coma
Nutrition	Very good	Good	Poor
Score	5-6	7-9	10-15

P.T.	Increase up to 3	Increase bet 3-6	Increase >6
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(Pugh's modification)

Palpable Left Lobe of the Liver

It lies in the epigastric region; its upper border cannot be felt; It moves with respiration; It extends towards left hypochondriac region; It is dull on percussion.

MASS IN THE EPIGASTRIUM

Parietal swellings

Sebaceous cyst, lipoma, neurofibroma, cold abscess (from ribs or spine, presents as soft, fluctuant nontender well localised swelling), liver abscess or subphrenic abscess rupturing into the abdominal wall presenting as parietal wall abscess.

Abscess in left lobe of liver rupturing into parietal wall

Epigastric hernia – specific

Intra-abdominal mass (Figs 21.43A and B and 21.44)

Left lobe of the liver – Abscess, hepatoma, secondaries

Stomach – Congenital pyloric stenosis, carcinoma, gastric ulcer perforation forming an abscess in the lesser sac, carcinoma of stomach, leiomyoma of stomach

Transverse colon mass

Omental mass

Pancreatic mass

Lymph nodal mass

Aortic aneurysm

Retroperitoneal swellings like cyst, sarcoma, teratoma

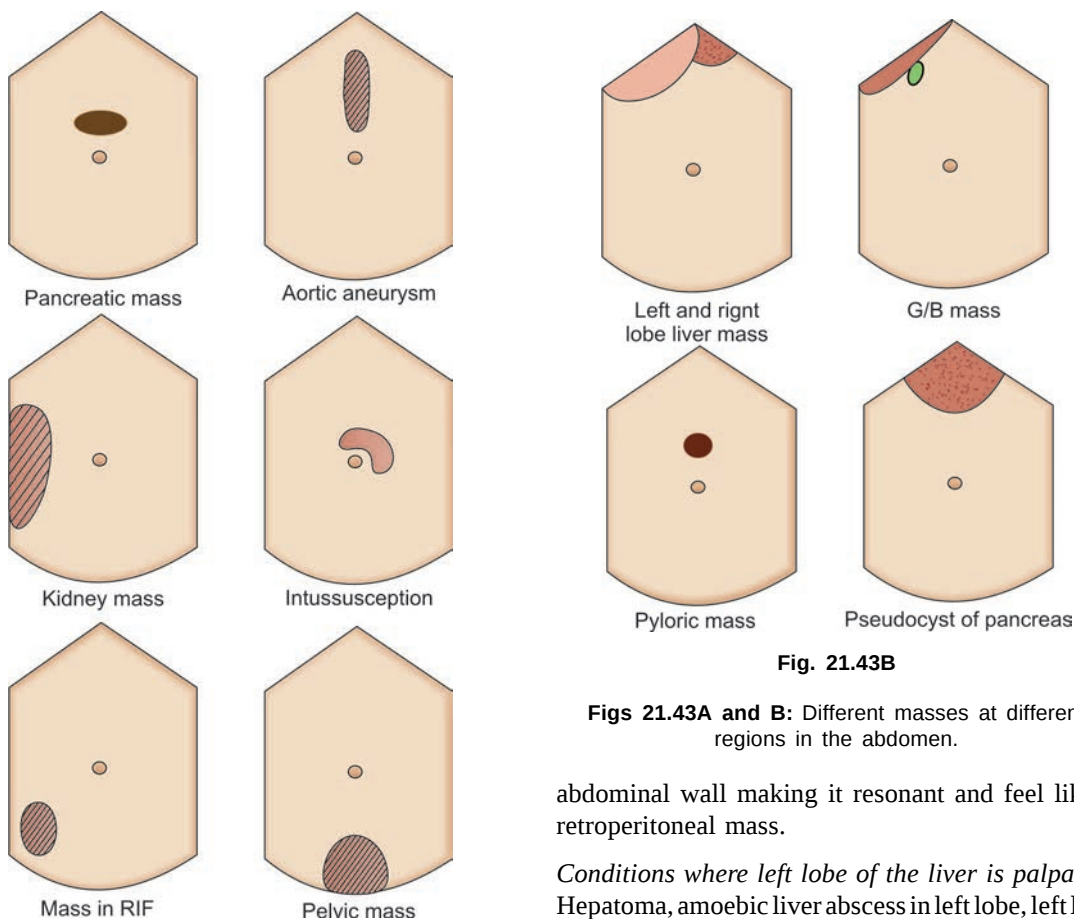


Fig. 21.43A

Fig. 21.43B

Figs 21.43A and B: Different masses at different regions in the abdomen.

abdominal wall making it resonant and feel like a retroperitoneal mass.

Conditions where left lobe of the liver is palpable: Hepatoma, amoebic liver abscess in left lobe, left lobe secondaries, hydatid cyst of the left lobe.

When amoebic liver abscess occurs in left lobe, it gets adherent to anterior abdominal wall, mass will not move with respiration and is immobile, causes oedema of the abdominal wall and is dull on percussion. Occasionally bowel may interpose between liver and

Features of Stomach Mass

It lies in the epigastric region; It moves with respiration; It is intra-abdominal; It is resonant or impaired resonant on percussion; Mass may be better felt on standing or on walking; Mass is often mobile, unless it gets

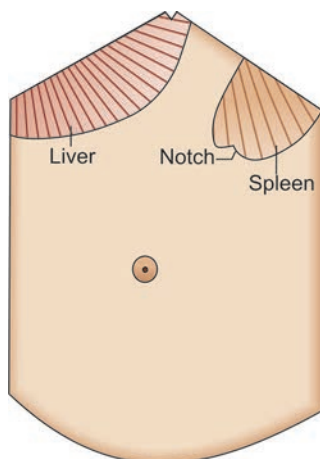


Fig. 21.44: Hepatosplenomegaly is the common condition (clinical entity). It is due to macronodular cirrhosis with portal hypertension, lymphoma, autoimmune diseases, congestive cardiac failure, hepatoma with portal hypertension, haemolytic diseases, etc. There may be ascites, supraclavicular palpable lymph node, pleural effusion (right sided).

adherent posteriorly; In pylorus mass, all margins are well felt which is mobile with features of gastric outlet obstruction; Mass from the body of the stomach is horizontally placed without any features of obstruction; Mass from the upper part of the stomach near the O-G junction causes dysphagia. Mass from the fundus of the stomach is in the upper part of the epigastric region towards left side. Carcinoma stomach is nodular and hard. It is commonest cause for stomach mass. Leiomyoma of stomach is smooth and firm.

Pseudocyst of the Pancreas

Mass lies in the epigastric region which is smooth, soft, does not move with respiration, not mobile, resonant on percussion. It can be tender if it gets infected, has got transmitted pulsation. It is confirmed by placing the patient in knee-elbow position. Lower border is well felt but upper border is not clear.

Baid test: Because stomach is pushed forwards, Ryle's tube when passed, can be felt per abdomen on palpation.

Pseudocyst of the pancreas is quite common condition. It has got a false capsule not true capsule as there is no epithelial lining. It usually occurs in 3 weeks after an attack of acute pancreatitis. Lesser sac is the common site. It also can occur in relation

Management of gastric carcinoma

Early growth—pylorus—lower radical gastrectomy with removal of tumour, proximal 5 cm clearance, nodal clearance, greater and lesser omentum, distal pancreas and spleen (now not regularly removed; it is removed to clear splenic nodes—one of the node stations) and Billroth II anastomosis or Roux-en-Y anastomosis is done. Postoperatively adjuvant chemotherapy should be given—5 fluorouracil, mitomycin, epirubicin, cisplatin. Growth in body, proximal growth, diffuse carcinoma and generalised linitis plastica are the indications for total radical gastrectomy with oesophagojejunal anastomosis. Neoadjuvant chemotherapy in advanced gastric cancer prior to surgery and later gastrectomy. Instillation of mitomycin C impregnated charcoal intra-peritoneally to control lymphatic disease (Japan). Palliative procedures like palliative partial gastrectomy, anterior gastroduodenostomy, Devine's exclusion gastrectomy, luminal stenting in proximal inoperable growths, chemotherapy are used in inoperable cases. In early carcinoma proper lymph nodal clearance is important.

Lymph node stations in gastric carcinoma—(Japan)—18 stations are there

1. Right cardiac
2. Left cardiac
3. Nodes along the lesser curvature
4. Nodes along the greater curvature
 - a. Along short gastric vessels—4sa
 - b. Along left gastroepiploic vessels—4sb
 - c. Along right gastroepiploic vessels—4sd
5. Suprapyloric nodes
6. Subpyloric nodes
7. Along left gastric artery
8. Along common hepatic artery
9. Along celiac axis
10. At splenic hilum
11. Along splenic artery
12. At hepatoduodenal ligament
13. Retroduodenal lymph nodes
14. At root of mesentery
15. Around middle colic artery
16. Para-aortic nodes
17. Around lower oesophagus
18. Supradiaphragmatic

to duodenum, jejunum, splenic hilum and colon. It can be of communicating and noncommunicating type. It often mimics aortic aneurysm, retroperitoneal cystic tumours, cystadenocarcinoma of pancreas.

Cystadenocarcinoma of the Pancreas

Mass is smooth, firm, does not move with respiration, not mobile, resonant on percussion. Patient also has back pain (**Figs 21.46A to C**).

Investigations for pseudocyst of pancreas— Ultrasound—commonly done procedure CT scan ideal and of choice (Fig. 21.45) LFT, serum amylase, prothrombin time ERCP to find out communications Barium meal—not done now—shows widened vertebro gastric angle	Indications for intervention— Size more than 6 cm Formed thick walled pseudocyst Infected pseudocyst
Interventions— Roux-en-Y cystojejunostomy is ideal Cystogastrostomy—Jurasz procedure—commonly done Cystoduodenostomy Cystogastrostomy with external drainage if infected—Smith operation Endoscopic stenting Laparoscopic cystogastrostomy—popular—safer Guided aspiration helps but high recurrence rate of 70%	Complications Rupture—3% Infection—20% Bleeding—torrential 7% Cholangitis
Acute fluid collection—just fluid collection Acute pseudocyst with thin wall Chronic pseudocyst—thick walled Pseudopseudocyst—inflammatory mass of bowel, omentum, etc. after acute pancreatitis mimics pseudocyst	



Fig. 21.45: Pseudocyst of pancreas—
CT scan picture

Colonic Mass

It is commonly due to carcinoma of transverse colon but can be due to intussusception, hyperplastic tuberculosis, and diverticulitis. It is mobile, horizontally placed, nodular, hard mass which does not move with respiration; Caecum will be dilated and palpable; It is resonant or impaired resonant on percussion; Patient will be having bowel symptoms, loss of appetite and decreased weight as seen in tuberculosis, carcinoma. Ileocolic intussusception is the commonest type

Para-aortic Lymph Node Mass

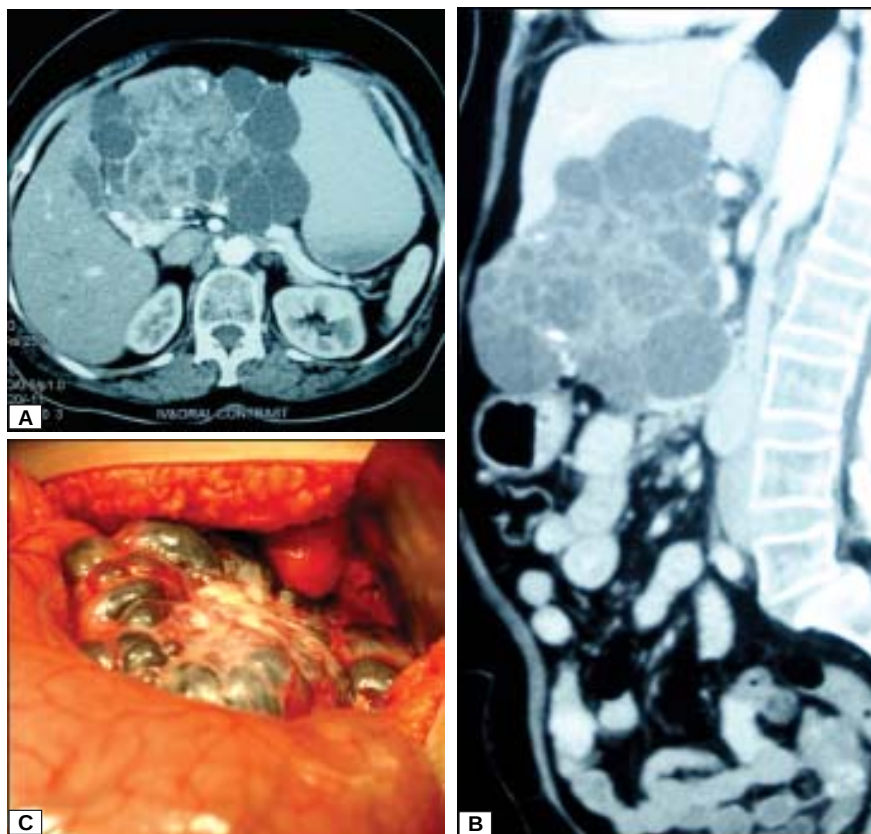
Presents as mass in the epigastric region which is deeply placed, not mobile, not moving with respiration. It is vertically placed, *level of the umbilicus* and resonant on percussion. Causes for enlargement are: *Secondaries, Lymphomas or Tuberculosis.*

Aortic Aneurysm

It is smooth, soft, pulsatile (expansile pulsation which is confirmed by placing the patient in knee-elbow position or in lateral position. Pulsation persists even in knee-elbow position; whereas transmitted pulsation disappears or decreases in intensity). It is vertically placed above the level of the umbilicus, not mobile, not moving with respiration and resonant on percussion.

Omental Mass

Omentum gets thickened with nodules and irregular surface. Omental mass moves with respiration, has nodular surface, firm in consistency, dull on percussion. Often lower margin is rolled up which is a feature of tuberculosis (Rolling is due to fibrosis). Omentum may get involved in malignancy as secondaries or in inflammatory conditions as part of inflammatory mass. Omentum is the usual component of any composite mass.



Figs 21.46A to C: Cystadenocarcinoma of pancreas from body and tail of pancreas—large extensive tumour.

Intussusception Ileocolic type is the commonest Red currant jelly Mass appears and disappears Smooth, firm sausage shaped mass around the umbilicus with concavity towards umbilicus Empty right iliac fossa Mass contracts under the palpating fingers	Carcinoma transverse colon Anaemia, loss of appetite and weight Alternate constipation and diarrhoea Palpable mass in the epigastrium or umbilical region (upper part)—nodular, hard, impaired resonant, does not move with respiration, mobile in all directions Features of obstruction/closed loop obstruction when ileocaecal valve is incompetent Ascites, liver secondaries later Colonoscopy proves the diagnosis
Colonic tuberculosis Usually hyperplastic type Loss of appetite and weight Irregular mass often adherent and nonmobile Impaired resonant Ascites, doughy abdomen may be present Difficult to differentiate clinically from carcinoma Colonoscopy confirms the condition	Inflammatory conditions like diverticulitis Pain, bowel symptoms Mass which is tender, firm, nonmobile Often it is a composite mass lesion comprised of small bowel, omentum May be adherent to abdominal wall Pericolic abscess, internal fistula may be the presentation

MASS IN THE LEFT HYPOCHONDRUM**Parietal swellings**

Sebaceous cyst, lipoma, neurofibroma, cold abscess (from ribs or spine, presents as soft, fluctuant nontender well localised swelling), liver abscess or subphrenic abscess rupturing into the abdominal wall presenting as parietal wall abscess

Intra-abdominal swellings

Splenomegaly—Malaria, Kala azar, hereditary splenomegaly, ITP, haemolytic anaemias, lymphoma, porphyria, splenic cysts (rare)
 Splenic flexure of colon—carcinoma
 Tail of the pancreas—pseudocyst, tumour
 Left subphrenic space
 Left kidney—hydronephrosis, RCC, polycystic disease
 Left adrenal gland tumour

Enlarged Spleen

Spleen has to enlarge three times to be palpated clinically. It enlarges towards the right iliac fossa from left costal margin. It moves with respiration, mobile, obliquely placed, smooth, soft or firm, with a notch on the superior margin near anterior end. Fingers can not be insinuated over the upper border. It enlarges downwards, inwards and forwards. '*Hook sign*' is positive, i.e. one cannot insinuate the fingers under the left costal margin. It is dull on percussion.

Left sided colonic mass (splenic flexure): It is mobile, nodular, resonant, and does not move with respiration. It is commonly due to carcinoma colon. Bowel symptoms like diarrhoea, tenesmus, constipation, intestinal obstruction, may be a feature.

Left renal mass from upper pole of any cause: It has got features of renal mass.

Left sided adrenal mass: It does not move with respiration. It is deeply placed mass, not mobile. Often it crosses the midline. It is resonant on percussion. It mimics kidney mass.

Mass Arising from the Tail of the Pancreas

It could be pseudocyst or cystadenoma/cystadenocarcinoma of pancreas. It is deeply placed mass, does not move with respiration, nonmobile, resonant.

Hereditary Spherocytosis

It is an autosomal dominant disease effecting males and females equally. Here there is an increase in red cell permeability to sodium. So sodium leaks into the red cells by which it becomes spherical and more fragile. This leads to greater loss of membrane phospholipid resulting in weakening of the membrane with increase in energy and oxygen requirement. So these RBC's are destroyed in spleen causing haemolytic anaemia, haemolytic jaundice, unconjugated hyperbilirubinaemia, pigmented gallstones, cholangitis.

Clinical features: Pallor, jaundice, recurrent fever, pain abdomen, splenomegaly, hepatomegaly, chronic leg ulcer. Gallstones are seen in 60% cases. Acute haemolytic crisis can occur.

Investigations: *Fragility test:* Here increased fragility of the erythrocytes is the typical feature. Haemolysis occurs in 0.6% or in even stronger solutions. Reticulocyte count is increased significantly. Faecal urobilinogen is increased. Labelled radioactive chromium shows faster red cell destruction. US abdomen is done to look for gallstones, spleen, liver, CBD. Peripheral smear, haematocrit and LFT. Direct Coomb's test is negative.

Idiopathic Thrombocytopenic Purpura (ITP)

It is development of *antiplatelet antibodies*, which damage patient's own platelets.

Splenic mass

Enlarges towards umbilicus and right iliac fossa—
 inwards, forwards, outwards
 Moves with respiration—well
 Splenic notch on the superior border anterior end
 Smooth, firm, dull on percussion
 Felt on superficial palpation
 Renal angle is normal and resonant
 Not bimanually palpable nor ballotable
 Cannot insinuate/hook under left costal margin

Renal mass

Enlarges downwards towards left iliac fossa
 Moves with respiration—slightly
 No notch is felt
 Colonic band of resonance in front
 Felt on deep palpation
 Renal angle is full; dull on percussion; may be tender
 Bimanually palpable and ballotable
 Can insinuate fingers under left costal margin

Clinical features: Purpuric patches in skin (buttocks and limbs), mucous membrane (most common presenting sign); epistaxis; menorrhagia, haematuria; GIT bleeding; intracranial haemorrhage (most dangerous); splenomegaly (25%); *Hess tourniquet test* is positive (By applying sphygmomanometer and inflating for 10 minutes just below the systolic pressure causes more than 20 petechiae in cubital fossa in 3 cm circled area).

Differential diagnosis: Other causes for purpura; increased capillary fragility; bone marrow suppression due to aplastic anaemia; chemotherapy; DIC; autoimmune diseases.

Investigations: Bleeding time is increased; Clotting and prothrombin time are normal; Platelet count is decreased; Bone marrow biopsy reveals increased megakaryocytes; US shows splenomegaly only in 25% cases.

Types: *Acute* is common in children. *Chronic* is common in adult. In children spontaneous regression occurs in 75% of cases after one attack.

Felty's syndrome

Chronic rheumatoid arthritis; leucopenia; splenomegaly, recurrent infections, ulcers in leg and ankles, anorexia, lymphadenopathy are the features.

Palpable Kidney Mass

There will be fullness in the loin which is better observed in sitting position. Mass moves with respiration. It is vertically placed, bimanually palpable, and ballotable. Renal angle is dull on percussion (normally it is resonant due to colon). There is a band of resonance in front due to reflected colon. It does not cross the midline.

Conditions where kidney gets enlarged:

Hydronephrosis: It is smooth, soft, lobulated, nontender mass.

Pyonephrosis: History of throbbing pain in the loin, pyuria and fever with chills. It is smooth, soft and tender kidney mass which is nonmobile due to inflammatory adhesion.

Perinephric abscess: Bulge in the loin; dullness on percussion; bending the trunk away from the side of the lesion; fever, tachycardia. *Mathe's sign:* In IVU, imaging in standing and lying down positions show kidney to be in same position whereas in normal individual kidney will be lower in standing position than in lying down position.

Polycystic kidney: History of loin pain and haematuria. Present with hypertension, anaemia and features of renal failure. Usually bilateral but one side presents early than the other side. It has lobulated, smooth surface.

Renal cell carcinoma: History of mass in the loin, haematuria, fever and dull pain. Mass is nodular and hard. It does not cross the midline.

Mass from the Ascending Colon on Right Side or Descending Colon on Left Side

History of altered bowel habits with decreased appetite and weight. Mass is nodular, hard which does not move with respiration and is not ballotable. It is resonant or impaired resonant on percussion. Renal angle is resonant. Proximal dilated bowel may be palpable.

Adrenal Mass

It is nodular and hard, does not move with respiration, not mobile and often crosses the midline. It is felt on deep palpation, resonant in front and not ballotable.

Retroperitoneal Tumours

They are not mobile, resonant and do not fall forward in knee-elbow position. They are deeply placed mass which are usually smooth and hard. They may be retroperitoneal sarcomas or teratomas, etc. Often

MASS IN THE LUMBAR REGION

Parietal wall swellings

Cold abscess in lumbar region may be due to Pott's disease
Lumbar hernia—impulse on coughing
Soft tissue tumours like anywhere

Intra-abdominal swellings

Renal mass
Liver mass right side; splenic mass left side
Colonic mass
Gallbladder mass right side
Retroperitoneal masses

MASS IN THE UMBILICAL REGION

Parietal swellings Umbilical adenoma Umbilical hernia rectus sheath haematoma, abdominal wall abscess common in this region Desmoid tumour	Intra-abdominal swellings Stomach and duodenum Small intestine—tumour, intussusception, inflammatory Mesenteric mass—cyst, tumour, nodal mass Transverse colon Omentum Pancreas Para-aortic nodes Aorta and iliac arteries Retroperitoneal swellings
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retroperitoneal tumours attain large size. IVC compression is often seen causing dilated veins in the lateral abdominal wall with direction of blood flow upwards. They occupy many regions in the abdomen. Obstruction of ureters can cause hydronephrosis (**Fig. 21.47**).



Fig. 21.47: Retroperitoneal tumour

Retroperitoneal Cysts

They are *smooth and soft* with the same features as retroperitoneal tumours. They often attain large size.

Usual masses are:

Mesenteric cyst; Omental cyst; Ovarian cyst (Pedunculated); Small bowel tumours; Extension of masses from other region.

Mesenteric Cyst

Causes: Chylolymphatic; Enterogenous; Cysts of urogenital remnant; Teratomatous dermoid cysts; Other

causes - Traumatic haematoma and cyst formation, Tuberculous cold abscess of mesentery, Hydatid cyst of mesentery.

Chylolymphatic cysts are the commonest one. It arises from congenitally misplaced lymphatic system. Common in ileum, is a thin walled cyst with flat endothelium, containing lymph or chyle which is either milky or cream coloured. It is solitary and commonly unilocular with loop of the bowel in front. It has got independent blood supply, i.e. not from the adjacent bowel loop. So enucleation is done without resecting bowel.

Enterogenous type arises as a diverticulum or duplication from the adjacent bowel. Hence it is a thick walled cyst (contains all layers of the bowel) and receives its blood supply from the adjacent bowel (not independent). So resection of the adjacent bowel along with the cyst is essential. Enucleation is contraindicated.

Clinical features: It is common in 2nd decade, often in childhood. It presents as painless abdominal swelling in umbilical region, smooth, fluctuant, not moving with respiration. It is mobile freely in the direction perpendicular to the line of mesentery. Line of attachment of the mesentery is an oblique line starting from a point 2.5 cm left of the midline and 1.0 cm below the transpyloric line extending downwards to the right iliac fossa at the junction of right lateral and transtubercular plane. There is a band of resonance in front of the cyst (**Figs 21.48 and 21.49**).

Complications of mesenteric cysts: *Torsion of cyst* can lead to *volvulus* of the adjacent bowel; *Rupture of the cyst*; *Haemorrhage* into the cyst; *Infection* - Patient presents with acute painful swelling in umbilical region.



Fig. 21.48: Mesenteric cyst.

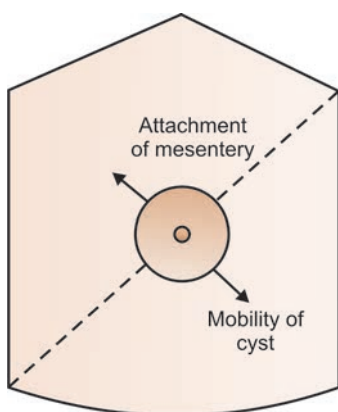


Fig. 21.49: Mobility of mesenteric cyst. Line of attachment of the mesentery is an oblique line starting from a point 2.5 cm left of the midline and 1.0 cm below the transpyloric line extending downwards to the right iliac fossa. It extends from ligament of Treitz at the level of L2 vertebra to right sacroiliac joint. Small bowel is 6 meters in length but mesentery is only 15 cm in length at its root.

Differential diagnosis: Hydronephrosis; Omental cysts; Tuberculosis.

Tillaux's triad

Soft fluctuant umbilical swelling
Freely mobile in a direction perpendicular to mesentery
Zone of resonance all around

Omental Cyst

It is smooth, soft and nontender, moves with respiration, mobile in all directions, dull on percussion. Omentum may also get involved by tuberculosis (rolled up omentum), secondary deposits (irregular and hard), may form a composite mass.

Small Bowel Swellings

Small bowel lymphomas; Small bowel tumours/ carcinomas; Intussusception.

Intussusception

Present as a mass in umbilical region usually towards left and above the umbilicus; occasionally towards right side. Mass is intra-abdominal, *sausage shaped*, well defined, smooth, firm and mobile. Mass does not move with respiration, contracts under palpating fingers. Often mass disappears and later reappears. Mass is resonant or impaired resonant on percussion. 'Red currant jelly' stool with features of intestinal obstruction may be present.

Tuberculous Mesenteric Lymphadenitis (*Tabes mesenterica*)

Matted lymph nodes of mesentery with coils of intestines can present as mass in umbilical region.

Diseases of the umbilicus

(1) Inflammations: Omphalitis; Umbilical granuloma; Pilonidal sinus. **(2) Fistulas:** (a) *Faecal*—Patent vitello intestinal duct, Neoplastic ulceration; Tuberculosis of peritoneum. (b) *Urinary*—Patent urachus. **(3) Neoplasms:** a) *Benign*—Adenoma (Raspberry tumour); Endometrioma. (b) *Malignant*—Primary (rare). Secondary carcinoma—Sister Joseph's nodule through lymphatics of the round ligament, primary being in the stomach, colon, ovary, uterus, breast (often blood spread). **(4) Umbilical hernias.** **(5) Umbilical calculus (Umbolith).**

Umbilical Granuloma

It is due to chronic infection of the umbilical cicatrix, causing sprouting of granulation tissue, leading to the formation of umbilical granuloma. It occurs in any age group, but common in infants and children. Present as umbilical discharge with tender, red swelling protruding from the umbilicus which bleeds on touch. It has to be differentiated from the anomalies of vitello-intestinal duct. It also mimics umbilical adenoma (Fig. 21.50).

Anomalies of Vitello-intestinal Duct

(1) It may remain completely patent, forming an *intestinal fistula*. (2) Only a small portion near the

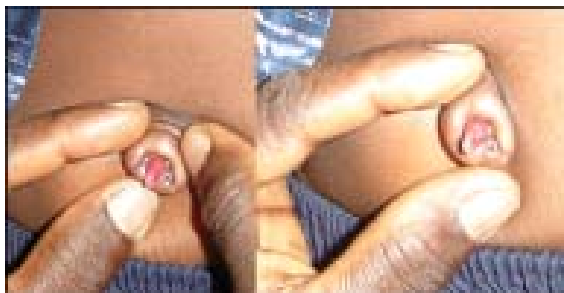


Fig. 21.50: Umbilical granuloma.

umbilicus may remain patent causing discharging *umbilical sinus*. Often the mucosa of this retained portion (epithelial lining) protrudes or everts to form *umbilical adenoma*. (3) Duct is closed on either side, but the intervening portion may remain as an *intra-abdominal cyst*. (4) Vitello-intestinal duct which is obliterated can retain as *band* which may be a seat for intestinal obstruction, volvulus, internal herniation. (5) *Meckel's diverticulum* itself can cause diverticulitis, obstruction. Fistulogram is useful. MRI delineates track well (**Figs 21.51 and 21.52**).

Umbilical Adenoma (Raspberry Tumour)

It is commonly seen in infants. It is due to partially obliterated vitello-intestinal duct towards umbilical side causing *prolapse of the mucosa* giving rise to *Raspberry tumour*, also called as *umbilical adenoma*.

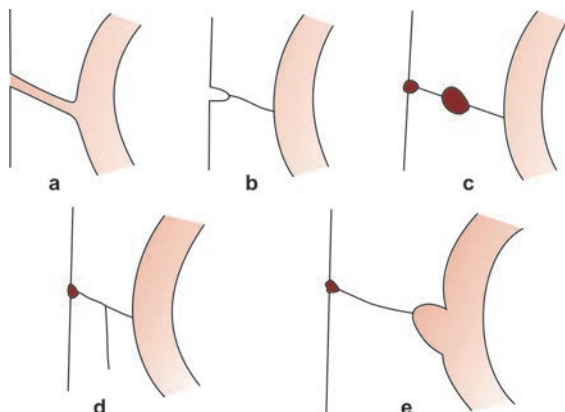


Fig. 21.51: Anomalies of vitellointestinal duct: (a) Intestinal fistula, (b) Umbilical sinus, (c) Intra-abdominal cyst, (d) Band, (e) Meckel's diverticulum.



Fig. 21.52: Patent vitellointestinal duct.

It protrudes out as a red swelling which is moist with mucus and tends to bleed on touch. It often gets infected, discharging pus through the umbilicus. Histologically, it consists of columnar epithelium rich in goblet cells (**Fig. 21.53**).

Differential diagnosis: Umbilical granuloma.



Fig. 21.53: Umbilical adenoma (Raspberry tumour).

Patent Urachus

Allantoic duct/stalk which is remnant of cranial part of ventral urogenital sinus forms urachus. It gets fibrosed and forms median umbilical ligament. When

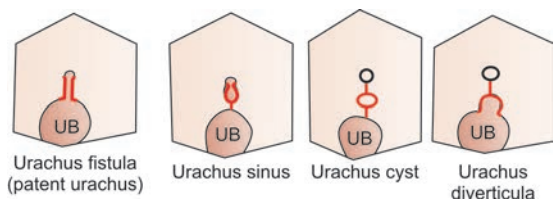


Fig. 21.54: Different types of urachal anomalies.

urachus is patent it can form—*Patent urachus (Urachal fistula)* between umbilicus and dome of the urinary bladder. *Urachal sinus occurs* when only umbilical side of the urachus remains patent. *Urachal cyst* occurs if only middle portion of the urachus remain patent with lining and fluid content. *Urachal diverticulum* occurs when bladder side of the urachus is patent (**Fig. 21.54**).

Features: Persistent discharge from the umbilicus often stained with urine if it is fistula; Recurrent infection and bleeding; Pain in the umbilicus and below; Recurrent urinary infection.

Investigations: Fistulogram to see the extent; US abdomen; Discharge analysis and culture; Urine analysis.

Umbilical Sinus

It is discharging sinus through umbilicus. It is common condition.

Causes: Persistent vitello intestinal duct towards umbilical side; persistent urachus; tuberculosis; umbilical infection or umbolith; pilonidal sinus of umbilicus; urachal malignancy.

Pain, swelling, discharge and tenderness over the umbilicus are the features. Discharge study—culture, cytology, AFB; sinusogram; CT abdomen; chest X-ray—needed investigations.

Umbilical Fistula

It is fistulous communication between umbilicus and organs in the abdomen either intestine or urinary bladder.

Causes: Patent vitello intestinal duct discharging faecal matter through umbilicus; patent urachus discharging urine; post-laparotomy; tuberculosis of abdomen either intestine or urinary bladder.

Along with discharge, pain, tenderness, excoriation is common.

Abdominal Wall Tumours

They are not uncommon but often present late as they are usually asymptomatic; Common tumours are lipoma, fibromas, neurofibromas, and fibromatosis. Malignant tumours occasionally when occurs, are either from skin or soft tissues. They may be desmoid tumour, soft tissue sarcoma like fibrosarcoma, dermatofibrosarcoma, liposarcoma, umbilical secondaries (*Sister Joseph Mary tumour*). Presentation is usually as painless progressive swelling. Often ulceration can occur. Attaining large size is also known. It is dull to percuss. On contracting the abdominal wall muscles swelling becomes prominent and less mobile. Differential diagnoses are—abdominal wall abscess, haematoma, intra-abdominal tumours (adherent to abdominal wall). US abdomen, CT abdomen is diagnostic. Biopsy is essential (**Fig. 21.55**).



Fig. 21.55: Abdominal wall large tumour. It may be Fibromatosis arising from abdominal wall.

Desmoid Tumour

It is a tumour arising from the musculoaponeurotic layer of abdomen, below the level of the umbilicus.

It is unencapsulated, hard, fibroma, presently classified under *aggressive fibromatosis*. 80% of cases occur in women, commonly after deliveries. It is common over old abdominal surgical scars (lower abdomen) may be due to old haematomas. It is often associated with the Familial polyposis colon (FAP), osteomas, odontomes, epidermal cysts (**Gardner's syndrome**). It is a slow growing tumour involving muscle and soft tissue of the abdominal wall, locally spreading, often undergoes myxomatous changes. Recurrence rate is high.

Exomphalos (Omphalocele)

It is the failure of all or a part of the gut to return to the coelomic cavity during early foetal life as coelomic cavity has not developed properly. Sac covering the content is very thin, consists of three layers—outer amniotic membrane, middle Wharton's jelly and inner peritoneal layer. Sac may get ruptured during birth.

Types: Two types:

Exomphalos minor: Here the sac is small and umbilical cord is attached to the summit, with small bowel as the content (**Fig. 21.56**).

Exomphalos major: A large defect is present with contents lying completely outside. Umbilical cord is attached to the inferior aspect of the sac. Contents are small bowel, large bowel and liver. Often the sac will rupture during delivery, which in turn leads to severe infection and high mortality. Here immediate surgery (within hours) is the only hope to save the



Fig. 21.56: Exomphalos minor.

life of the child. Omphalocele is often associated with the *congenital anomalies of the cardiac and genitourinary system* (**Fig. 21.57**).



Fig. 21.57: Exomphalos major. Note the liver, small and large bowel.

Gastroschisis (Belly Cleft)

It is a defect of the anterior abdominal wall just lateral to the umbilicus. It is common in premature babies. Umbilicus is normal. The defect is almost always to the right of an intact umbilical cord. Evisceration of the bowel develops through the defect during intrauterine life. There is no peritoneal sac and irritating effect of amniotic fluid causes chemical peritonitis with formation of thick, oedematous membrane. Non-rotation and intestinal atresia are common associations. Cardiac anomaly is not common as in omphalocele. After delivery, these infants are more prone for fluid loss, hypothermia, hypovolaemia, sepsis, metabolic acidosis. Necrotising enterocolitis is also common in such infants (20%). They are also more prone for paralytic ileus (**Fig. 21.58**).

Rectus Sheath Haematoma

Rectus abdominis muscle is supplied by superior and inferior epigastric arteries. Injury to one of these vessels will cause bleeding and haematoma in rectus sheath. Commonly it is due to bleeding from inferior epigastric artery in the lower abdomen.



Fig. 21.58: Gastroschisis.

Causes: Trauma; surgery; spontaneous haematoma; blood dyscrasias; severe straining and exercises; tetanus and other convulsions; patients on anticoagulants; puerperium.

Features: Common in females; Sudden onset of swelling in lower abdomen, which is tender, warm, firm on one side of the abdomen. Swelling does not cross the midline; bluish discolouration is seen over the swelling; US and aspiration confirms the diagnosis; should be differentiated from other masses and parietal hernias.

Abdominal Wall Abscess

Causes: Infected haematoma; Umbilical sepsis spreading into the abdominal layers causing the abscess; Blood spread from distant focus.

Features: Tender, soft/firm, nonmobile swelling which is well localised, adherent to skin and underlying abdominal muscles. Aspiration will show pus. It should be ruled out from intra-abdominal mass, cold abscess, parietal hernia. US is diagnostic (**Fig. 21.59**).



Fig. 21.59: Abdominal wall abscess.

Meleney's Progressive Synergistic Bacterial Gangrene of the Abdominal Wall

It is due to infection by microaerophilic streptococci, staphylococci and other anaerobes of the postoperative abdominal or thoracic wounds. It is common in HIV, diabetic and immunosuppressed people. Sudden pain, redness, blackening and gangrene of the skin of the abdomen with abdominal wall necrosis. Toxicity, septicæmia, renal failure can occur

Appendicular Mass

It is *smooth or granular, firm, tender* mass in the right iliac fossa. It is formed by dilated ileum, omentum, inflammatory fluid and inflamed appendix which is often adherent to the abdominal wall. It is not mobile. It does not move with respiration. It is *resonant on percussion* (**Figs 21.60 A and B and 21.61**). It is well localised mass with distinct borders. It develops 3-4 days after an attack of acute appendicitis. Commonly with conservative treatment (*Ochsner Sherren* regime)

MASS IN THE RIGHT ILIAC FOSSA

Parietal swellings	Intra-abdominal swellings
Abdominal wall tumour	Appendicular mass or abscess
Abdominal wall abscess	Carcinoma caecum
Iliac abscess, appendicular abscess extending into the abdominal wall	Ileocaecal tuberculosis
Actinomycosis in right iliac fossa often extending into the abdominal wall and may form discharging sinuses with sulphur granules	Amoeboma
	Psoas abscess
	Lymph node mass either mesenteric or external iliac lymph nodes
	Bony swellings
	Ectopic kidney
	Undescended testis (Abdominal)
	Actinomycosis



Figs 21.60A and B: Mass in the right iliac fossa.



Fig. 21.61: Percussion over the mass in right iliac fossa. Retroperitoneal mass is resonant. Bowel mass is impaired resonant. Mass from abdominal wall is dull on percussion.

mass gradually reduces in size. Size of the mass should be marked out to observe the daily response. Occasionally if they don't respond for therapy, sepsis

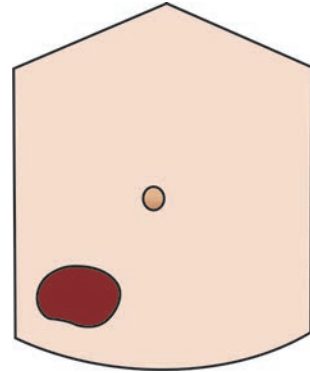


Fig. 21.62: Appendicular mass—location.

may progress leading to peritonitis or suppuration may occur causing appendicular abscess (**Fig. 21.62**).

Appendicular Abscess

It is smooth, *soft, tender and dull mass* in the right iliac fossa with indistinct borders. It is located on lower and outer aspect of the right iliac fossa. As pus has got tendency to come to surface in dependent position, it reflects the bowel towards periphery and so it is dull on percussion. Redness and abdominal wall oedema is evident. Appendicular abscess need to be drained surgically (**Fig. 21.63**).

Carcinoma Caecum

It is nodular, hard mass in the right iliac fossa. It does not move with respiration. It is mobile but mobility may be restricted once it gets adherent to psoas major muscle behind. Mass is resonant or impaired resonant on percussion. Anaemia, anorexia, loss of weight is

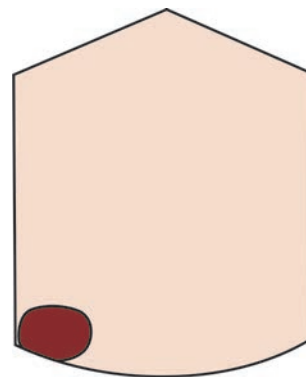


Fig. 21.63: Appendicular abscess—location.

common. Occasionally features of intestinal obstruction may be present. Carcinoma with pericolic abscess may present as tender firm smooth mass in the right iliac fossa. Fever, tachycardia are also the features. Barium enema X-ray/colonoscopy/carcinoembryonic antigen (CEA)/US abdomen are the investigations.

Ileocaecal Tuberculosis

Mass in the right iliac fossa which is smooth, hard, resonant and nontender; does not move with respiration and has restricted mobility; Caecum may be *pulled up* to lumbar region due to fibrosis. It is often clinically difficult to differentiate from carcinoma and ileocaecal tuberculosis. Bowel symptoms, anaemia with loss of appetite and weight is common. Barium studies, colonoscopy, CT abdomen are the investigations needed. Obstruction may be the presenting feature. Usually hyperplastic type of ileocaecal tuberculosis present as mass in right iliac fossa.

Amoeboma

History of dysentery with pain in the right iliac fossa may be present. Well defined palpable mass in the right iliac fossa which is smooth, hard, not mobile, may or may not be tender. It slowly increases in size and after certain period it stops progression. Initially features of amoebic typhilitis may be present. *Amoebic typhilitis* is inflammation of caecum due to *Entamoeba histolytica* infection. Tenderness over both iliac regions with thickening of colon is common. Amoebic typhilitis is usually associated with sigmoid amoebic colitis. Perforation, bleeding, stricture, paracolic abscess formation, ischiorectal abscess and fistula formation are the complications.

Crohn's Disease or Regional Ileitis

It is a *granulomatous, noncaseating inflammatory condition of the ileum commonly and of the colon often*.

Aetiology: Unknown, but a familial and infective nature is thought of. Diet, food allergy, mycobacterium paratuberculosis are thought of.

Pathology: Inflammation → Granuloma formation → Cicatrification → Thickening of the bowel wall → Adhesions → Fistula formation. Mesentery is

thickened, oedematous, with enlarged lymph glands which will never break nor calcify. Rarely jejunum, stomach and other parts of GIT are involved. In colon, it is commonly observed in caecum and ascending colon. Anal fissure is very common association.

Clinical features: (a) *Acute presentations (5%)* of Crohn's disease mimics acute appendicitis with severe diarrhoea. Often there will be localised or diffuse peritonitis. (b) *Chronic Crohn's: First stage:* Mild diarrhoea, colicky pain, fever and tender, firm, non-mobile mass in right iliac fossa with recurrent perianal abscess. Anaemia and diarrhoea is usual. *Second stage:* is either acute or chronic intestinal obstruction due to cicatrification with narrowing. Steatorrhoea, colitis, anaemia, fissure in ano, fistula in ano is common. *Third stage:* Fistula formation—enterocolic, enteroenteric, enterovesical, enterocutaneous, etc. Crohn's disease is independent of age, sex, social and economic status and geographic area.

It is familial. **It is precancerous condition but not as much as ulcerative colitis.**

Investigations: Barium meal follow through shows: Straightening of valvulae conniventes; Multiple defects (*cobblestone* appearance); Cicatrification of ileum (*string sign of Kantor*); *Rose thorn appearance of the bowel wall*. Radiologically Crohn's disease is classified as **nonstenosing type or stenosing type**.

Actinomycosis of Right Iliac Fossa

Disease begins in caecum, inflammatory mass develops which gets adherent to abdominal wall in right iliac fossa. Mass will be nonmobile irregular hard, often tender due to secondary infection. Later induration of abdominal wall develops followed by suppuration and multiple discharging sinus formation discharging sulphur granules. Disease process is often triggered by appendectomy.

Roundworm Bolus Mass in Right Iliac Fossa

It presents as smooth, soft or firm, yielding rounded mass in the right iliac fossa which is mobile and tender due to adjacent enteritis. Features of intestinal obstruction—distension, vomiting, constipation, ill health, malnutrition are evident. It is common in children; common in developing countries.

Iliac Lymph Node Mass

Iliac nodes are located in the right iliac fossa on medial aspect above the inguinal ligament. It is deeply seated mass which is smooth/nodular, firm or hard. If it is of inflammatory origin it may be smooth and firm or soft and tender. In lymphoma it is smooth and firm; nodular and hard in secondaries.

Mesenteric Lymph Node Mass in Right Iliac Fossa

It may be due to tuberculosis, lymphoma, secondaries or composite mass.

Ilio Psoas Abscess

It is localised; smooth, soft, nonmobile mass in the right/left iliac fossa. *Psoas spasm* (flexion of the hip joint) is typical. Spine may show *gibbus*, *tenderness*, *paraspinal spasm*. Spinal movements will be restricted. Tuberculosis of sacroiliac joint also can cause cold abscess. Often psoas abscess extends below the inguinal ligament lateral to the femoral artery. Such patient develops swellings on either sides of the inguinal ligament which is cross fluctuant.

Ectopic Kidney

It is a developmental abnormality wherein kidney does not ascend to its normal position. Ectopic kidney may be in the pelvis or in the right iliac fossa. It is deeply placed firm nonmobile mass in the right iliac fossa which does not move with respiration. It is resonant on percussion. Usually when it is pathological it is palpable like hydronephrosis, pyonephrosis, polycystic kidney disease, or neoplastic disease. IVU is diagnostic. Radioisotope scan is done to see the function. CT is also needed.

Undescended Testis

Testis from lumbar region descends to scrotum along the inguinal canal. Failure of descent makes it imperfectly/undescended testis. It may be abdominal or inguinal in location. Abdominal testis may be in right iliac fossa. It is often difficult to palpate and identify as it is usually atrophied. These undescended testes are 20 times more prone for malignant transformation and when it develops, it may be clinically palpable as mass in right iliac fossa (left iliac fossa in left side) which is nodular, hard, nonmobile.

Mobile Kidney

It is usually normal kidney which attains undue mobility probably having peritoneal covering also which can be brought down far as below as to right iliac fossa in right side type. But kidney can be replaced back to normal location.

Hydrops Gallbladder

Enormously distended gallbladder can descend down and may palpable in right iliac fossa.

Pelvic Masses

Ovarian tumour/cyst; tuboovarian mass; uterine fibroid; pyosalpinx; broad ligament cyst can present as mass in iliac fossa. Lower border of such mass merges into the pelvis and so is not felt; on per vaginal examination mass is well felt. It is bimanually palpable often done under general anaesthesia. Emptying the bladder is important while examining the pelvic masses.

Urinary Bladder Diverticulum

It can be felt as a soft, tender, and mobile mass in the iliac fossa which may get emptied partially after catheterisation. Cystogram, cystoscopy and CT abdomen confirms the diagnosis.

Mass in the Left Iliac Fossa

All conditions are same as in right iliac fossa. Appendicular mass and abscess will not occur here. Sigmoid pathology—diverticulitis and carcinoma are left iliac fossa diseases.

Diverticular Disease of the Colon

They are *acquired herniations* of colonic mucosa through circular muscles at the points where blood vessels penetrate. It is commonly localised to sigmoid colon (90%) but occasionally seen in full length of the colon. It is a *false diverticulum* with only *mucosal* herniation (**Fig. 21.64**). Rectum is *not* affected. *Saint's triad* (5%) is diverticulitis; hiatus hernia; gallstones. It is rare in Asian and African countries because of the high fibre diet. It is common in Western countries. **Diverticulosis** is the initial primary stage of the disease wherein there is hypertrophy, muscular incoordination

MASS IN THE HYPOGASTRUM

Parietal swellings Urachal cyst Abdominal wall abscess Abdominal wall tumours like in other regions	Intra-abdominal swellings From urinary bladder From uterus, Fallopian tube and ovaries—fibroid, ovarian cyst, tubo-ovarian mass Pelvic abscess Tumours of pelvic bone—chondrosarcoma Pelvic soft tissue mass
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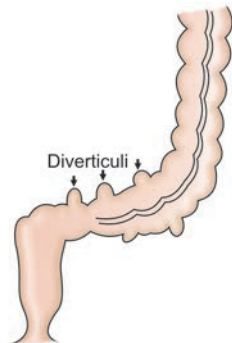


Fig. 21.64: Diverticular disease of colon.

leading to increased segmentation and increased intraluminal pressure. At this stage they are often asymptomatic, but very often get severe spasmodic pains due to colonic segmentation, fullness of abdomen, bloating and flatulent dyspepsia called as *painful diverticular disease*. **Diverticulitis** is the second stage due to inflammation of one or more diverticula with pericolicitis. It presents with persistent pain in left iliac fossa, lower abdomen distension, bleeding per anum, fever, loose stool, recurrent constipation, tenderness in right iliac fossa, palpable and thickened sigmoid colon. Mass may be palpable in the left iliac fossa which is smooth, soft, tender, nonmobile because of inflammatory adhesions. Often abdominal wall oedema with redness may be present. P/R may reveal a tender mass. It is the *commonest cause* of lower GI bleed in *Western countries*.

Complications of diverticulitis: Perforation and pericolic abscess or peritonitis; progressive stenosis and intestinal obstruction; Profuse colonic haemorrhage (17-20%); Fistula formation (5%)—colovesical (commonest type with pneumaturia occasionally passing faeces); colovaginal; coloenteric; colocutaneous.

Note: Diverticulitis is not a pre-cancerous condition.

Investigations: Barium enema shows 'saw-teeth' appearance/*champagne bottle sign*. Sigmoidoscopy is useful but should not be done in acute stage. Once acute stage subsides, barium enema, sigmoidoscopy, colonoscopy can be done (to rule out only *associated malignancy*). Carcinoma/amoebic proctitis/tuberculosis are differential diagnosis.

Carcinoma of Sigmoid Colon

It presents as discomfort, fullness in left iliac fossa with diarrhoea, constipation, tenesmus, bleeding per anum, colonic obstruction. Often a hard, nodular mass may be felt in the left iliac fossa, initially mobile but later becomes immobile once it is fixed. It can often be soft and tender if there is complication of pericolic abscess. In such occasion it may be adherent to anterior abdominal wall.

Bladder Mass

It is in the lower midline. Lower abdomen is distended which is more obvious on standing. It is dull on percussion. Lower border is not felt. It can be mobile in horizontal direction. Mass reduces in size after emptying the bladder. It can be felt on per-rectal examination. All causes of retention of urine cause palpable bladder. It also can be neoplastic either carcinoma bladder (common) or leiomyoma or sarcoma bladder.

Uterine Mass

It is midline mass which is smooth or hard. Lower border extends into the pelvis and is not felt. Pregnancy with history of amenorrhoea has to be elicited. History of last menstrual period is important (LMP); History of vomiting, lower abdominal discomfort is common; urine pregnancy test and US confirms pregnancy.

Uterine fibroid is the commonest tumour which is felt per abdomen in the midline or often extending into iliac fossae. It is slowly progressive, vertically placed, horizontally mobile, firm nodular mass, lower border is not felt as it is merging into pelvis, dull on percussion, ascites is not a feature. It is felt on pervaginal examination. Occasionally leiomyosarcoma or endometrial sarcoma may be the cause of uterine mass. They are smooth, firm often soft, rapidly progressive mass in the hypochondrium.

Ovarian Mass

It is smooth, soft, tensely cystic, mobile mass merging into the pelvis, felt per vaginally. It should be differentiated from ascites. Ascites is dull in the flank, resonant in the centre/summit of the abdomen; ovarian cyst is dull in the centre, resonant in the flanks as intestines are pushed towards periphery. *Blaxland (Athelstan Blaxland) ruler test* shows pulsation in ovarian cyst not in ascites (**Fig. 21.65**).

In all lower abdomen masses P/R and/or P/V is must. Bladder should be emptied using a catheter prior to palpation (**Fig. 21.66**).

Investigations for Mass Abdomen

Haematocrit, Liver function tests, renal function tests, stool/urine examination.

Ultrasound abdomen.

Endoscopies—Gastroscopy-Colonoscopy-ERCP-MRCP.

Barium studies—Barium meal-Barium enema- Barium meal follow through.

CT scan—contrast CT is ideal for mass abdomen as it clearly gives idea about the origin of mass, its extent and operability, vascularity, relation to major vessels. Intravenous as well as oral water soluble iodine contrast agent should be given (**Fig. 21.67**).

MRI.

Endosonography.

Ascitic tap.

Diagnostic laparoscopy.

US guided/ CT guided biopsy.

IVU/RGP/Cystoscopy/Isotope renogram.

Exploratory laparotomy.

In all regions parietal masses can occur

Benign and malignant soft tissue tumours. Commonest is lipoma.

Fatty hernia of linea alba, interstitial hernia.

Desmoid tumour.

Parietal wall abscess.



Fig. 21.65: Blaxland ruler test to feel pulsation.



Fig. 21.66: Ovarian cyst—large tumour on table finding.



Fig. 21.67: CT scan abdomen showing ascites and secondaries in liver.

22

Examination in Rectal and Vaginal Problems

Rectal examination is an essential part of the surgical field without which clinical methods in surgery is incomplete.

History

History of Present Illness

Bleeding Per Rectum

This is the most important history and commonest history to which patient attends surgical clinic. Quantity of blood loss; nature of blood (colour); whether frank blood or not; relation with faeces; duration of blood loss—are important. Bright red means bleeding is from rectum and anal canal; dark red means it is from the colon proximal to rectum; black or altered blood means from small bowel; melaena means from upper gastrointestinal bleed. Bleeding from proximal colon causes blood to mix with faeces as the soft faeces of proximal colon can mix well with blood. Bleeding from the anorectum causes blood on the surface of the stool. Blood separate from faeces occurs when blood collects in the rectum, irritates it, causing urge to defecate and patient will pass blood with mucous; it is seen in carcinoma of rectum, diverticulitis, polyps, and ulcerative colitis. Fresh blood in the pan or toilet paper at the end of defecation is due to bleeding from

piles or fissure. Mucous discharge is common in complete prolapse. Bleeding in children is due to rectal polyp (**Fig. 22.1**).



Fig. 22.1: Prolapsed pile – 3rd degree – 7 O'clock position showing both internal and external component. It is the common cause of bleeding per rectum.

History of Discharge

Discharge may be purulent, mucous or blood mixed. It can be due to fistula in ano, piles, colitis, carcinoma, Crohn's disease. There may be foul smelling, severe discharge as seen in carcinoma of rectum.

History of Pain

Pain in the anus: Disease below the dentate line is painful; above it is painless. Throbbing pain is seen in perianal abscess (with swelling); sharp severe pain is seen in acute fissure in ano; pain beginning at the time of defecation and persists with burning even after some time is a feature of chronic fissure in ano. Fistula

Bright red colour	Polyps
Red currant jelly	Intussusception
Maroon coloured stool	Meckel's diverticulum
Blood mixed with stool	Carcinoma of colon
Blood streaked on stool	Carcinoma of rectum
Blood and mucous	Colitis
Blood only	Diverticulitis, carcinoma of rectum
	Piles
Blood after defecation/ blood splashes in the pan	Upper GI bleed
Melaena	

in ano presents with intermittent pain, swelling that becomes severe, and opens at the point where it regularly discharges, pus come out; discharge develops. Such episode repeats at regular intervals and patient presumes that fistula has healed. Once discharge starts it is relatively painless. Piles when complicated become painful (thrombosis, prolapse, infection, strangulation). Carcinoma of rectum initially is painless but become painful once it infiltrates pararectal tissues and nerve plexus/spreads below the dentate line.

Pain in the abdomen: It is probably due to intestinal obstruction may be subacute or acute due to an annular sided growth. Here inflammation (colitis) causes oedema and blocks the lumen completely. Spasmodic pain abdomen is common in lower abdomen in colorectal diseases like carcinoma, ulcerative colitis.

History of Altered Bowel Habits

Constipation, diarrhoea, alternate constipation and diarrhoea, spurious diarrhoea, tenesmus are different presentation of altered bowel habits. Left sided stenosing growth causes constipation. Stasis of faecal matter causes infection and colitis leading into mucous discharge distally across stenosed area to cause diarrhoea. Annular type presents rather early due to constipation and so carries good prognosis. If there is a proliferative growth in the rectal ampulla, then patient has *incomplete sense of evacuation* after defecation due to sensation of fullness in rectum. Because of this sensation of fullness, patient strains painfully and tries to empty the bowel but without succeeding which is called as *tenesmus*. In ulcerative type of carcinoma rectum, overnight collected faecal matter in the rectum which also contains blood, mucous, pus from ulcerated lesion irritates and stimulates the rectal wall, causing in the patient real urgency to pass stool once he gets up in the morning—‘*morning spurious diarrhoea*’. *Tape-like/pipestem*

stool is a feature of anorectal growth due to narrowing of the passage.

History of Mass per Anum

Mass coming out through the anus during defecation may be due to rectal polyp, haemorrhoids, rectal prolapse. Mass which has come out may retract back to original position spontaneously or by manual push (using fingers). In 4th degree piles, one cannot push the pile mass outside. In complete prolapse often it cannot be pushed back inside and is called as *proctidentia*.

History of Itching/Pruritus (Latin) Ani

Itching in perianal region is common and is due to many causes. Often it is intractable. It may be due to causes in *anorectum* (piles, fissure, etc); causes in the *vagina* (*Trichomonas*, *Candida*, gonorrhoea, cervical erosions); *perianal skin* conditions (*Tinea cruris*, *Candida*); *parastitic* causes (threadworm); *general* causes (psychogenic, lack of hygiene). Pruritus may be *wet* (fissure, fistula, carcinoma, over intake of liquid paraffin) or *dry* (diabetes, poor hygiene).

History of loss of weight and reduced appetite is seen in carcinoma, ulcerative colitis, diverticulitis.

Past History

Previous history of surgery for piles, fistula, anorectal abscess; history of tuberculosis, drug treatment (for ulcerative colitis, Crohn’s) are important.

Family History

Haemorrhoids, polyps, carcinoma often run in family.

Examination of the Anorectum

‘If you don’t put your finger in (to the rectum), you put your foot in it’.

Causes of pruritus ani –

Poor hygiene	Allergy/dermatitis/psoriasis
Fissure/fistula/piles	Intertrigo
Warts/polyps	Cervical erosions
<i>Trichomonas vaginalis</i> infection in females	Erythrasma (<i>Corynebacterium minutissimum</i>)
Candidiasis, gonorrhoea, tinea	Diabetes mellitus
Parasites (threadworm)/epidermophytosis	Psychological causes
Carcinoma anorectum	

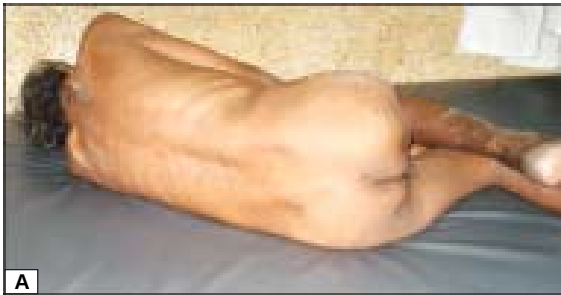
It is *criminal negligence* if digital examination of rectum is not done in an anorectal case.

10 cm of the anus/anorectum can be assessed by digital examination.

First *inspection of the anal canal* is done; then *palpation of the anal canal*; later *digital examination* of the rectum is done.

Different Positions Used

Left lateral–Sims' position: Patient lies with right leg placed above the left and flexed; left leg below the right and semiflexed, buttocks projecting over the edge and trunk across the bed (not parallel) (**Figs 22.2A and B**).



Figs 22.2A and B: Sims' position and doing per rectal examination.

Right lateral position: It is the position with right leg up and flexed to feel the rectosigmoid growths which will fall forward and downward (**Fig. 22.3**).

Dorsal position: It is used in severely ill patients where changing the position of the patient is contraindicated. Patient lies on the bed supine in semi-recumbent



Fig. 22.3: Digital examination in right lateral position.

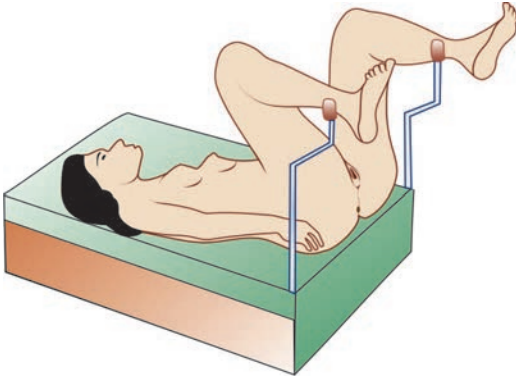
position, with hips and knees slightly flexed. Right hand of the examiner is passed behind the right thigh of the patient and anorectum is felt using right index finger. Left hand of the examiner is kept over the suprapubic region for bimanual palpation. Rectovesical pouch and pelvis assessment can be done well in this position but anus cannot be inspected in this position (**Fig. 22.4**).



Fig. 22.4: Digital examination of the rectum in dorsal position.

Lithotomy position: It needs special table to position the patient. This position is suitable for bimanual examination under general anaesthesia, proper inspection of the anus and palpation. Biopsy and therapeutic procedures can be carried out in this position (**Fig. 22.5**).

Knee-elbow position: It is used in palpation of prostate and seminal vesicle. Seminal vesicles are normally

**A****B**

Figs 22.5A and B: Lithotomy position used for examination and all perineal surgery like for fissure, piles, and fistula, abdominoperineal resection for carcinoma rectum (APR).

so soft and not palpable. It becomes palpable in tuberculous seminal vesiculitis, *Trichomonas vaginalis* infestation, abacterial nongonococcal urethritis (**Fig. 22.6**).



Fig. 22.6: Rectal examination in knee-elbow position.

Picker position: It is used to palpate seminal vesicles in obese patients or in patients with prostatic hyperplasia. Patient stands and leans forward grasping a low chair or stool; seminal vesicles are palpated using fingers (**Fig. 22.7**).



Fig. 22.7: Picker position to feel seminal vesicles.

Inspection of the Anal Canal and Perineal Area

Sentinel pile, fissure, external opening of fistula, pilonidal sinus, condyloma, anal carcinoma, perianal inflammation are inspected. Fissure in ano is checked by retracting the buttocks laterally using fingers on each side. It is common in posterior midline position. Location, number of the openings in fistula in ano should be noted. Orifice looks like a whitish raised small point area with often redness around and discharge from the orifice. Distance of the external orifice from the anus should be noted. Posterior fistulas are curved and anterior fistulas are straight/direct (in the same line radial direction) is the common rule called as *Goodsall's rule*. Often there may be multiple external openings. *Pilonidal sinus* is confirmed in prone position retracting the buttocks properly. Small sinus opening or openings are evident in the midline and paramedian positions few centimeters proximal to the anus over the sacrum posteriorly in midline.

Condyloma acuminata are multiple, pedunculated papilla like lesions caused by papilloma variant virus. It is transmitted by sexual contacts. Itching, discomfort, pain, ulceration due to rubbing against clothes is common features. They may be associated with other sexually transmitted diseases like HIV, gonorrhoea. It may involve wider area of the perineum, labia majora, scrotum, etc. *Condyloma lata* occurs in secondary syphilis as flat raised white hypertrophied epithelium at mucocutaneous junction. It is highly contagious. Ulcer with everted edge with bloody discharge is feature of anal carcinoma. Often it may be proliferative lesion. Itching marks with redness and oedema around may be a feature of inflammation. Prolapse rectum is examined with patient in *squatting position* and he is asked to strain as if he is passing the stool (**Fig. 22.8**). Rectal mucosa protruding out downwards can be seen. If it is less than 3 cm it is partial prolapse; if it is more it is complete (**Figs 22.9 and 22.10**). Prolapsed piles should be differentiated from prolapsed rectum. A prolapsed pile is segmental with a skin cover over it and mucosal part on the inner aspect. Rectal polyp or intussusception of the colon also rarely may protrude out through the anal canal. Pigmented *melanoma* lesion may be evident if carefully inspected. It may be mistaken for thrombosed piles. Ulcerated *melanoma* in the anus may not be black (**Figs 22.8 to 22.22**).

Piles	Rectal prolapse
Segmental	Circumferential
Plum coloured	Red coloured
Outer cover of skin with inner mucosa	Only mucosa

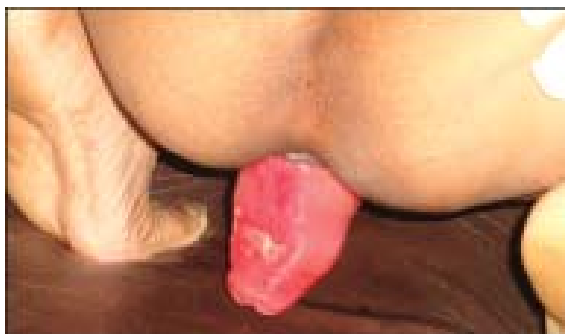


Fig. 22.8: Complete rectal prolapse (procidentia) in squatting position with a decubitus ulcer on it.



Fig. 22.9: Partial rectal prolapse is less than 3.75 cm and only mucosal.



Fig. 22.10: Complete rectal prolapse. It should be confirmed by observing the patient during straining in squatting position.



Fig. 22.11: Typical external pile. It can cause haematoma, abscess, pain, itching.



Fig. 22.12: Multiple fistulas in ano. It may be Crohn's disease; carcinoma or HIV. Biopsy should be taken prior to formal therapy. MRI of perineum helps a lot in such patients to anatomically evaluate the fistula in ano.

Palpation of the Perineum and Anal Canal

Perineum should be palpated for abscess, perianal haematoma, ulcer, growth, external opening of the fistula in ano. Tenderness, induration, palpation of the edge of an ulcer for induration (anal canal carcinoma) should be done. Ischiorectal abscess will be tender and indurated. In rectal prolapse it should be differentiated from *intussusception (sigmoidorectal)*.

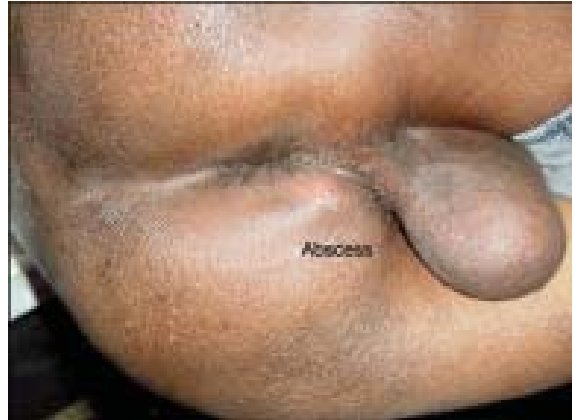


Fig. 22.13: Perianal abscess. Note the swelling and inflammation.



Fig. 22.14: Parts of piles—plum coloured internal part and black cutaneous external component. It is 1st degree piles.



Fig. 22.15: Anatomical locations of internal and external piles. It is 2nd degree piles.



Fig. 22.16: Thrombosed prolapsed pile. Note the colour of the thrombosed pile. It is very painful and tender.



Fig. 22.19: By retracting the buttocks in prone position pilonidal sinus is inspected.



Fig. 22.17: Prolapsed, strangulated piles. It is 4th degree piles.



Fig. 22.20: Anal papilloma. It often attains very large size. It could be of viral origin. It can turn into squamous cell carcinoma.



Fig. 22.18: Multiple fissures in ano. Note the method of retraction of the buttock. Note the prominent posterior fissure with sentinel pile.



Fig. 22.21: Anal canal carcinoma. Squamous cell carcinoma is commonest type – 80%.



Fig. 22.22: Large rectal polyp which has come out of the anal canal.

In intussusception, finger can be insinuated between intussusceptum and anal canal; but in rectal prolapse finger cannot be insinuated between prolapsed mucosa and skin margin. *Bidigital palpation* of the anal canal with one finger inside, thumb outside and feeling the wall should be done in abscess or fistula. Fistula track may be *multiple*. Specific causes like tuberculosis, carcinoma, lymphogranuloma venereum, bilharziasis, Crohn's disease, ulcerative proctitis should be thought of. Fistula due to *tuberculosis* shows clear watery discharge, ragged margin, discoloured surrounding skin *without* any protrusion or induration (**Figs 22.23 and 22.24A and B**).



Fig. 22.23: Perianal haematoma.



Figs 22.24A and B: Bidigital examination (palpation) of the anal canal.

Digital Examination of Anorectum (Per Rectal; P/R)

It is *contraindicated* in acute fissure in ano as it causes severe pain.

Often enema is necessary prior to rectal examination to clear the *loaded faeces* in the rectum.

Proper positioning is done. Disposable gloves are used. Patient should be told about the procedure and asked to breathe through mouth and relax. Anus is

lubricated with a lubricant or xylocaine jelly. Pulp of the index finger is used. In children little finger may be used. Pulp is kept flat over the anus. With gentle pressure finger is pushed gradually into the anal canal once anal sphincter relaxes and gently rotated. Tenderness, sphincter tone, wall of the anal canal for thickening should be assessed. *Anal groove* or anal intermuscular depression is felt just inside the anal verge which is demarcation between external and internal haemorrhoidal plexus and external and internal sphincter muscles. *Anorectal ring* is situated at the junction of anal canal and rectum. It is 2-3 cm in length. Puborectalis component of levator ani muscle is arranged like a sling and so it is well felt laterally and behind it. Fistula or anorectal abscess should be assessed in relation to this ring. Finger is pushed as high as possible. *First lumen* is felt; *then wall* of the rectum; *lastly* deeper plane *outside* the rectal wall is felt (**Figs 22.25A and B and 22.26**).



Figs 22.25A and B: Correct method and wrong method of doing finger examination of the rectum. Pulp should be used parallel while passing the finger into the anal canal (not perpendicularly using tip of finger).



Fig. 22.26: Rectal examination in lithotomy position.

Digital examination of rectum (P/R)

No abdominal examination is complete without a per rectal examination

- A. It is done to palpate
 1. Carcinoma rectum
 2. Stricture rectum
 3. Polyps
 4. Thrombosed piles
 5. BPH and carcinoma prostate
 6. Secondaries in the rectovesical pouch (Blumer shelf).
 7. Sphincter tone
 8. Pelvic abscess (is felt as boggy swelling).
- B. To feel internal opening in anal fistulas
- C. In bimanual palpation of the bladder or pelvic tumours.
- D. In acute abdominal conditions—It reveals dilated empty rectum with tenderness.

Loss of sphincter tone is confirmed by giving digital traction on the sphincter or by hooking the finger around the anorectal ring or sphincter when gaped, rectal mucosa and lumen is displayed. Vaginal deliveries, badly performed rectal surgeries, congenital, neurological and *senility*/old age (most common cause) are some of the causative factors.

In the lumen:

Lumen of rectum is spacious. Ballooning of the rectum is a feature of intestinal obstruction. Mass may be felt. It is better felt during straining. Faecal matter often hard may be felt.

In the wall:

Lower valve of Houston may be felt in the mucosa like a rim. Ulceration, irregularity, proliferative lesion,

tenderness should be felt. Size, shape, induration, extent, number of lesions should be felt. Whether upper limit can be reached or not should be assessed.

Stricture type lesion whether due to trauma or post-operative or carcinoma or tuberculosis or lymphogranuloma venereum or chronic proctitis of any cause will be felt like narrowed area where finger may not be able to passed through it. Diaphragm like periphery with central hole is typical. A narrow crescentic circular mucosal fold felt 4 cm from anal verge in young individual is probably due to imperfect fusion of the hindgut to proctodeum causing *congenital stricture*. Postoperative, postradiotherapy and traumatic strictures are *fibrous* type. Malignant stricture is *hard, irregular and indurated* type. Stricture of Lymphogranuloma is *tubular and rubbery*. 90% of rectal carcinoma can be felt by per rectal finger examination. Mobility/fixity, extent of deeper infiltration should be analysed. **Bimanual examination** with other (left) hand over the suprapubic region should be done. Extent into the bladder, prostate or vagina in females should be assessed. Posterior spread into the sacrum should be confirmed. Internal opening of the fistula in ano can be felt as a button-like indurated area in *midline* (commonly) on posterior surface. Often it can be high or multiple or in anterior surface. *Thrombosed piles can be felt by finger* (usually internal piles cannot be felt as it is very soft; it is only seen through proctoscope). *Polyp* of rectum may be felt as a rounded soft swelling with warty irregular surface; its extent, base can also be determined; often it can be pulled down into the anus to properly inspect and feel.

Outside the rectal wall:

When finger is passed high above sacral promontory can be felt. Ischial spine can be felt laterally.

Prostate is felt anteriorly deep to mucosa. Both lateral lobes are felt. Normally it is bilobed, smooth, firm, rubbery with a central/median sulcus or groove with rectal mucosa moving freely over it. In benign prostatic hyperplasia (BPH), it is felt as smooth, firm enlarged prostate with median groove in the centre. In carcinoma prostate, it may be nodular and hard. Size is not a criteria for carcinoma prostate. In BPH, prostate may be large; in carcinoma it may be normal sized. Base of the bladder, rectovesical pouch, seminal vesicles are felt anteriorly. Seminal vesicles are felt

in knee-elbow position or *Picker* position. Seminal vesicles are felt in upper lateral aspect of the prostate. Secondaries in rectovesical pouch are felt in front above the prostate as a hard mass deep to mucosa and are called as *Blumer shelf* (George Blumer—New Haven Connecticut). Pelvic abscess is felt as smooth, soft, tender, and *boggy* swelling in front of this pouch.

In females, cervix, uterus, vagina and rectouterine pouch (Douglas) is felt per rectally. Cervix is felt in front like projection – *pons asinorum* (bridge of asses – Latin). Uterus also can be well felt bimanually. Retroverted uterus can be confirmed. If patient is wearing a pessary it can be felt like a mass. Blood, secondaries, pus can be felt in this pouch. Lump outside the rectal wall, subserosal fibroid, oedematous Fallopian tube can be felt.

Lateral palpation: Ischiorectal fossa, lateral wall of pelvis, lower end of ureters, internal iliac arteries are felt laterally. Ischiorectal abscess is felt as tender tense swelling. A stone in the lower ureter, iliac artery aneurysm may be felt through rectal examination. Pelvic bone mass (tumour or infective), hip joint central dislocation, fracture pelvis can be felt. Pelvic appendicitis causes tenderness and often mass in the lateral wall of rectum in front. Salpingitis, ovarian cyst/tumour, ectopic gestation can be felt per rectally.

Behind, coccyx can be palpated using index finger inside and thumb over coccyx for tenderness and abnormal movements of *coccydynia*. Sacrococcygeal teratoma and post-anal dermoid can also be felt.

Any pelvic mass can be felt well on rectal examination and bimanual examination. Size, shape, consistency can be assessed (**Fig. 22.27**).



Fig. 22.27: Bimanual palpation of the rectum.

Note:

Mucosal lesions are better felt by downwards stroking than upward pushing of the finger.

After finishing the rectal examination, finger should be inspected for presence of blood, faeces, mucous or pus. It can be collected in gauze for proper inspection.

Massive oedema of rectal wall may be noted in sigmoid volvulus due to inferior mesenteric vein occlusion.

Often it is difficult to find out whether lump felt is in the wall or outside. Finger is placed on one side of the lump and slid over the elevation of the lump to feel the presence or absence of continuity of the overlying mucosa. Pelvic masses, urinary bladder can be assessed well by bimanual rectal examination.

In female child per vaginal examination is usually avoided; per rectal examination allows palpation of entire pelvic viscera.

In neonates and infants up to 3 months, rectal examination is done using little finger.

Examinations

Examination of Lymph Nodes

Carcinoma below the dentate line spreads to horizontal group of inguinal lymph nodes; carcinoma above this line, spread to iliac nodes. Iliac nodes can be felt by deep palpation above and medial aspect of the inguinal ligament.

Abdominal Examination

It is done to look for liver secondaries (nodular hard liver); ascites, features of intestinal obstruction.

Vaginal Examination

Always change the glove after doing vaginal or rectal examination. Usually vaginal examination is done first then followed by rectal examination. 'Never insult the vagina by examining the rectum first'. Even then glove should be changed after vaginal, before rectal examination.

Position: Left lateral position is used to begin with. Dorsal position is better to examine the urethral orifice. Lithotomy position is needed for proper assessment and bimanual examination. General anaesthesia is preferred.

Inspection: Labia should be inspected for swelling, skin changes. Surrounding area should also be inspected. Ulcer, swellings, itch marks, introitus should be inspected. Presence or absence of hymen should

be noted in introitus. Small sharp edged opening admitting only the fingertip is a virgin hymen. In pregnancy introitus looks bluish. Blood stained discharge is seen in menstruation, abortion, ectopic pregnancy, and carcinoma. White purulent discharge is seen in vaginitis, cervicitis, endometritis and pelvic infections. Profuse watery, yellow often frothy discharge with pruritus is a feature of *Trichomonas vaginalis* infestation. Thick curdy discharge is seen in *Candida* infection. Purulent discharge is seen in gonorrhoea.

Palpation: Labia are separated using thumb and forefinger of the left hand. Using lubricated index and middle fingers of the right hand palpation is done. Index finger is introduced first then middle finger is passed. First cervix then anterior, lateral and posterior fornices are palpated in that order.

Chancre, lymphogranuloma venereum, granuloma inguinale, chancroid, herpes, leukoplakia, carcinoma, papillomas can occur in vulva. Sebaceous cyst also can occur in vulva. Pruritus marks may be evident. Vaginal discharge may be evident.

Bartholin glands are palpated over posterior part of the labia majora between finger and thumb. Gland is deep and posterior. Bartholin cyst is a retention cyst. It often can get infected forming an abscess. Often it is bilateral (**Fig. 22.28**).

Straining down will make cystocele obvious. Cystocele is descent of bladder through anterior vaginal



Fig. 22.28: Bartholin cyst forming an abscess.

wall. Rectocele is descent of rectum through posterior vaginal wall.

Stress incontinence is checked in full bladder by asking the patient to cough and urine will spill out from the bladder.

Anovaginal bidigital examination: It is done by placing index finger in the rectum and thumb in the vagina or by placing right index finger in the rectum and left index finger in the vagina. It is used to palpate anterior anorectal lesion or posterior vaginal wall lesion or to check the musculature of the perineum (tone).

Bimanual examination (Nicolas Puzos, Paris): Lubricated right hand fingers are kept high in the vagina; left hand fingers from above the pubic symphysis pressed downwards and backwards. Size, position (ante or retroverted) of the uterus is made out; in thin females ovaries may be felt. Normal Fallopian tubes are not palpable. When it is enlarged it may be palpable. Size, shape, extent, surface, consistency of the pelvic swelling is assessed. Relation of the bladder to the mass anteriorly and pouch of Douglas posteriorly is assessed.

Carcinoma cervix, carcinoma uterus, fibroid uterus, ovarian neoplasm, endometriosis should be considered in vaginal examination.

Proctoscopy (Kelly's)

Indications

Diagnostic—Piles, fissure in ano, polyps, stricture, etc. **Therapeutic**—Injection therapy for partial prolapse or piles, cryotherapy for piles, polypectomy, biopsy for carcinoma rectum or anorectum.

Types

Illuminating and nonilluminating.

Parts

Proctoscope is conical shape, with proximal diameter more than the distal, so as to illuminate the light at the required site properly. **Obturator** is the inner part which allows the easy insertion of the proctoscope.

Positions for Proctoscopy

Left lateral position (common), right lateral, lithotomy, knee-elbow position.

Technique of Proctoscopy

After doing digital examination, proctoscope with the obturator is introduced inside, through the anal canal in the direction towards the umbilicus. The obturator is removed. Proctoscope is withdrawn and during the course of withdrawal, any pathology has to be looked for. **Acute anal fissure is contraindication for proctoscopy (Figs 22.29 to 22.33).**

Sigmoidoscopy

It is used to visualise rectum and sigmoid colon, take biopsies from suspected lesions and do therapeutic procedures (polypectomy, control of bleeding, etc). There are two types: (1) **Rigid**—25 cm long, with illumination. (2) **Flexible**—60 cm long. In lateral position as in P/R examination or proctoscopy, sigmoidoscope with obturator is passed into the rectum and obturator is removed. Rectosigmoid is inflated with air and scope is negotiated into the sigmoid by **Alpha (α) manoeuvre**. Looked for any disease, biopsies are taken and also any required procedure is done. **Precaution:** Should be careful in acutely inflamed sigmoid colon, because chances of perforation is high (Fig. 22.34).



Fig. 22.29: Proctoscopic view of the internal pile.



A



B

Figs 22.30A and B: Proctoscopy and obturator. Grooved proctoscopy is also shown which is useful for therapeutic purpose.



Fig. 22.31: Proctoscopy used for therapy – cryosurgery for piles.



A



B

Figs 22.32A and B: Proctoscopy in knee-elbow position. It can be done in left lateral, lithotomy positions also.



Fig. 22.33: Anoscope and proctoscope. Anoscope is smaller than proctoscope.



Fig. 22.34: Rigid sigmoidoscope with inflation balloon and biopsy forceps.

Colonoscopy

It is 160 cm long, flexible scope. It helps to visualise full length of the colon; to take biopsies from different parts of the bowel; to identify synchronous growths; to remove polyps. Technique is same as sigmoidoscopy, but can be passed up to the caecum. It takes a long time and requires expertise to do the same (**Fig. 22.35**).

Carcinoma Rectum

It is common in females. It usually originates from a pre-existing adenoma or papilloma (tubular polyp); in 3% of cases it occurs in multiple sites (synchronous).



Fig. 22.35: Colonoscopic view of polyp.

Aetiology: Red meat and saturated fatty acids increase the risk; High fibre diet reduces the risk; Alcohol and smoking increases the risk; FAP and adenomas are more prone to carcinomas.

Gross: It can be *Ulcerative; Papilliferous; Infiltrative*.

Histologically It is *adenocarcinoma*.

Spread: *Local spread*—Initially, it spreads locally circumferentially (takes 12-18 months to complete the circumference of the bowel). Later spreads out to the muscular coat and peri-rectal tissue; then to prostate, bladder, seminal vesicles in males and uterus and vagina in females; posteriorly into the sacrum and sacral plexus, laterally into the ureters. *Lymphatic spread:* Above the peritoneal reflection, spread occurs upwards along the colic lymph nodes. In midrectum into the para rectal and midrectal lymph nodes. Downward spread is rare, occurs when growth is close to the anal canal, into the inguinal lymph nodes. *Venous spread* occurs into the *liver, lungs, adrenals* and other areas.

Duke's staging of carcinoma rectum

- A. Confined to bowel wall, mucosa and submucosa.
- B. Extends across the bowel wall to the muscularis propria with no lymph nodes involved.
- C. Lymph nodes are involved.

Modified Duke's staging

- A. Growth limited to rectal wall.
- B. Growth extending into extra rectal tissues but no lymph node spread.
 - B1: Invading muscularis mucosa.
 - B2: Invading into or through the serosa.
- C. Lymph node secondaries.
- D. Distant spread to liver, lungs, bone, brain.

TNM staging of rectal cancers

- Tx Primary not assessed.
- T₀ No primary tumour.
- Tis Carcinoma in situ.
- T₁ Invasion to submucosa.
- T₂ Invasion to muscularis propria.
- T₃ Invasion of subserosa or non peritonealized perirectal tissues.
- T₄ Involvement of visceral peritoneum, other organs or structures.
- N₀ No nodal spread.
- N₁ 1-3 nodal spread.
- N₂ 4 or more nodal spread.
- M₀ No distant spread.
- M₁ Distant spread present.

Clinical Features

Bleeding per rectum/anum (may mimic haemorrhoids); morning spurious diarrhoea; tenesmus; bloody slime; sense of incomplete evacuation; altered bowel habits; urinary symptoms are due to infiltration of the bladder or prostate; back pain due to invasion of sacral plexus; ascites, liver secondaries, urinary symptoms. **90% of rectal growths can be felt by per-rectal examination.**

Investigations

Proctoscopy; sigmoidoscopy; biopsy using *Yeoman's* forceps; barium enema in case of FAP and synchronous growths; US abdomen; CT scan to see operability; MRI pelvis; *endorectal ultrasonography* (Figs 22.36 and 22.37).



Fig. 22.36: CT scan showing carcinoma of rectum.



Fig. 22.37: MRI showing carcinoma of rectum and adjacent structures. MRI is good method to evaluate the spread and staging.

Differential Diagnosis

Inflammatory stricture, amoebic granuloma, tuberculosis, carcinoid, solitary ulcer syndrome.

Rectal Prolapse

It is circumferential descent of bowel through the anal canal. It is commonly seen in infants, children and elderly individual. It is common in females (6:1).

Aetiology

Decreased sacral curvature and decreased anal canal tone are the probable causes in infants. Diarrhoea, cough, malnutrition are the additional factors in children. In adults, it is common in females, common in multiparas. It is due to weakening of supporting tissue and levator ani muscle, atony of the sphincter, increased intra-abdominal pressure due to any cause (like neurological diseases, spinal injury, old age).

Partial prolapse wherein only mucosa and submucosa of the rectum descends, not more than 3.75 cm. There is no descent of the muscular layer. It is the *commonest type of rectal prolapse*.

Clinical features: There is H/O mass coming per anum, which can be observed when child is allowed to strain in squatting position. It is *pink in colour and circumferential*.

Complete prolapse: It is also called as *proctidentia*, is *less common than partial prolapse*. It is common in females (6:1). It is due to weakened levator ani and supporting pelvic tissues. The descent is always more than 3.75 cm, contains all layers of the rectum (i.e. including muscular layer). Often descends down up to 10-15 cm. It is often associated with the uterine descent (*uterine prolapse*). It is also thought to be as an intussusception of the rectum.

Clinical features: Complete descent of the rectum which is red in colour, often painful; bleeding can occur because of the congestion; sepsis, discharge, fever, anaemia are other features. P/R examination shows lax sphincter. Anteriorly, peritoneal sac comes down as a pouch which may contain small bowel (Figs 22.38 and 22.39).

Differential diagnosis: Rectosigmoid intussusception, third degree piles.



Figs 22.38A and B: Complete rectal prolapse.



Fig. 22.39: Sigmoidorectal intussusception – coming out through anal canal.

Pilonidal sinus (jeep bottom; driver's bottom)
- Pilus - hair; Nidus - nest

It is of infective origin and occurs in sacral region between the buttocks, umbilicus and axilla. It is common in hair dressers (seen in interdigital clefts), jeep drivers. It is common in 20-30 years of age. It is common in males and mostly affects hairy men.

Commonest site: Interbuttock sacral region. Hair after penetrating the skin causes dermatitis, infection, pustule and sinus formation which again sucks hair further by negative pressure forming pus and granulation tissues leading into multiple primary and secondary sinuses. *Primary sinus* occurs in the midline. *Secondary sinus* occurs laterally (paramedian).

Clinical features: *Discharge* - either serosanguinous or purulent; *Pain* - throbbing and persistent type; *tender swelling* seen just above the coccyx in the midline (*primary sinus*); and on either sides of the midline (*secondary sinus*). Tuft of hairs may be seen in the opening of the sinus (Figs 22.40 and 22.41).

Causes for recurrence (20%): Improper removal; overlooked diverticulum of the sinus; entering of the new hairs through the scar; breaking of the scar.



Fig. 22.40: Pilonidal sinus – primary and secondary sinuses are clearly seen.



Fig. 22.41: Pilonidal sinus—operative jack knife position.

Piles/haemorrhoids/figs

Piles = a ball or mass, Haemorrhoids = blood to ooze, Figs = a fruit (Anjoora).

It is abnormal sliding downwards of anal cushions due to straining or other causes.

Types: Internal; External; Internoexternal.

Classification I: Primary haemorrhoids: Located at 3, 7, 11 O'clock positions, related to the branches of the superior haemorrhoidal vessel which divides on the right side into two, left side into one (**Fig. 22.42**).

Secondary haemorrhoids: One which occurs between the primary sites.

Classification II: First degree haemorrhoids—Piles within, that may bleed but do not prolapse.

Second degree haemorrhoids—Piles that prolapse during defecation but return back spontaneously.

Third degree haemorrhoids: Piles that prolapse during defecation but can be replaced only by manual help (**Fig. 22.43**).



Fig. 22.42: Position of primary haemorrhoids.



Fig. 22.43: Haemorrhoids 3rd degree.

Fourth degree haemorrhoids: Piles that are permanently prolapsed (**Fig. 22.44**).

Piles begin as pedicle and it is located at the origin of the internal pile, i.e. at the level of anorectum.

Aetiology

Hereditary; Morphological - weight of the blood column without valves causes high pressure. Veins in the lower rectum are in loose submucosal plane,



Fig. 22.44: Prolapsed pile – 4th degree.

but the veins above enter the muscular layer, which on contraction increases the venous congestion below (more prevalent in patients with constipation). Superior rectal veins have no valves (as they are tributaries of portal vein) and so more congestion. Other causes are straining, diarrhoea, constipation, overpurgation, carcinoma rectum, pregnancy; portal hypertension (rare cause).

An arterial pile: It is haemangiomatous condition of superior rectal artery entering the pedicle of internal haemorrhoid which will bleed profusely.

Clinical Features

Bleeding—1st symptom ‘Splash in the pan’—‘bright red and fresh’—occurs during defecation; **Mass per anum;** **Discharge**—a mucoid discharge; **Pruritus;** **Pain**—may be due to prolapse, infection or spasm, etc; **Anaemia;** On inspection, prolapsed piles will be visualised; On P/R examination only thrombosed piles can be felt. Through *proctoscopy*, exact position can be seen as a bulge into the proctoscope. Sigmoidoscopy or colonoscopy or barium enema should be done if there is any suspicion of associated malignancy.

Complications

1. Profuse haemorrhage which may require blood transfusion.
2. Strangulation—piles is being gripped by anal sphincter.
3. Thrombosis—piles appears dark purple / black, feels solid and tender.
4. Ulceration.
5. Gangrene.
6. Fibrosis.
7. Stenosis.
8. Suppuration leading on to perianal or submucosal abscess.
9. Pylephlebitis (Portal pyaemia) is rare but can occur in 3rd degree piles after surgery.

Causes for bleeding per anum

Piles	Carcinoma rectum
Fissure in ano	Carcinoma colon
Polyps	Diverticulitis
Ulcerative colitis	Intussusception
Amoebic colitis	Vascular anomaly of the colorectum
Fistula in ano	Mesenteric ischaemia

Anal Fissure (fissure in ano)

It is an ulcer in the longitudinal axis of the lower anal canal. Commonly it occurs in the *midline, posteriorly (more common in males)*, but can also occur in the midline anteriorly (more common in females).

Causes: Because of the curvature of the sacrum and rectum, hard faecal matter while passing down causes a tear in the anal valve leading to posterior anal fissure. Anterior anal fissure is common in females due to lack of support to pelvic floor. **Other causes**—Haemorrhoidectomy, Crohn’s disease, venereal disease, ulcerative colitis, tuberculosis, etc.

Types: Anal fissure can be *acute or chronic*. Fissure ends above at the *dentate line*. **Acute anal fissure:** It is a deep tear in the lower anal skin with severe sphincter spasm without oedema or inflammation. It presents with *severe pain and constipation*.

Chronic anal fissure: It has got inflamed, indurated margin with scar tissue. Ulcer at its inferior margin is having a skin tag which is oedematous, acts like a guard - *Sentinel pile*. Proximally *hypertrophied anal papilla* is seen. It can cause repeated infection—fibrosis—abscess formation—fistula formation. Chronic fissure is less painful than acute one.

Clinical features: Common in middle aged women, not in elderly. Pain is severe in nature in acute type

whereas less severe in chronic. Constipation, bleeding and discharge are other features; P/R examination and proctoscopy is *not possible* in acute fissure in ano. General anaesthesia is required for examination. In chronic fissure, ulcer is felt with button-like depression, induration and often sentinel pile (**Figs 22.45 and 22.46**).

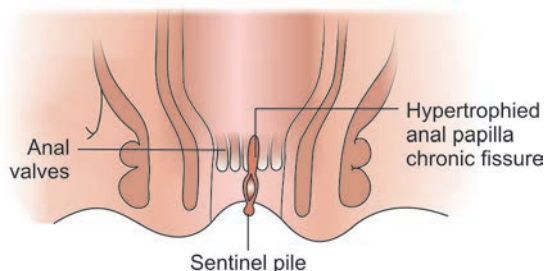


Fig. 22.45: Parts of chronic fissure in ano. Ulcer in the lower anal canal; sentinel pile below and hypertrophied papilla above.



Fig. 22.46: Fissure in ano (posterior). Note the ulcer with skin tag.

Differential diagnosis: Carcinoma anal canal; Inflammatory bowel disease; venereal diseases; anal chancre (painful); tuberculous ulcer; Proctalgia fugax.

Anorectal Abscess

Commonest causative organism is *E.coli*. Others are *Staph. aureus*, *Bacteroides*, *Streptococcus*, *B. proteus*. Common origin is by infection of an anal gland.

Other causes: Injury to anorectum; cutaneous infection; blood born infections. Many anorectal abscesses are associated with anal fistulas.

Classification: Perianal; Ischiorectal; Submucous; Pelvirectal; Fissure abscess (in relation to fissure in ano).

Perianal (60%): This usually results due to suppuration of anal gland or suppuration of thrombosed external pile or any infected perianal condition. It lies in the region of subcutaneous portion of external sphincter.

Clinical features: Pain in perianal region; tender smooth swelling in the region, with difficulty in sitting.

Ischiorectal abscess (30%): Commonly it is due to extension of low intermuscular anal abscess laterally through external sphincter. But often it can be blood or lymphatic born. Fat in the fossa is more prone for infection because it is least vascularised. Fossa communicates with that of opposite side through post-sphincteric space and so *horse-shoe like* abscess can occur. It presents with tender, indurated, brawny swelling in the skin over the ischiorectal fossa with high fever. *Well localised swelling and fluctuation are absent in ischiorectal abscess* (**Fig. 22.47 and 22.48**).

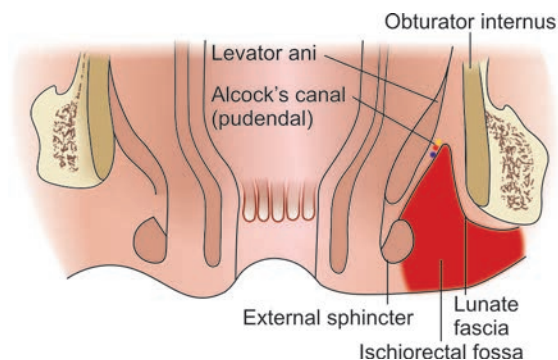


Fig. 22.47: Anatomy of ischiorectal fossa. Note the boundaries and Alcock's canal.

Submucous abscess (5%): It occurs above the dentate line, which can be drained with sinus forceps, through a proctoscope.

Pelvirectal abscess: It is situated between the upper surface of levator ani and pelvic peritoneum. It is almost like a pelvic abscess, occurs secondary to appendicitis,



Fig. 22.48: Ischiorectal abscess drainage under general anaesthesia. Note the cruciate incision placed and pus gushing out.

salpingitis, diverticulitis, Crohn's. US abdomen is done to rule out the above factors.

Differential diagnosis of anorectal abscess

Periurethral abscess (**Fig. 22.49**)
Bartholin abscess
Tuberculous abscess

Problems with anorectal abscess

Recurrent abscess formation
Fistula formation



Fig. 22.49: Periurethral abscess is a differential diagnosis for perianal abscess. Catheterisation is a must before drainage of an abscess.

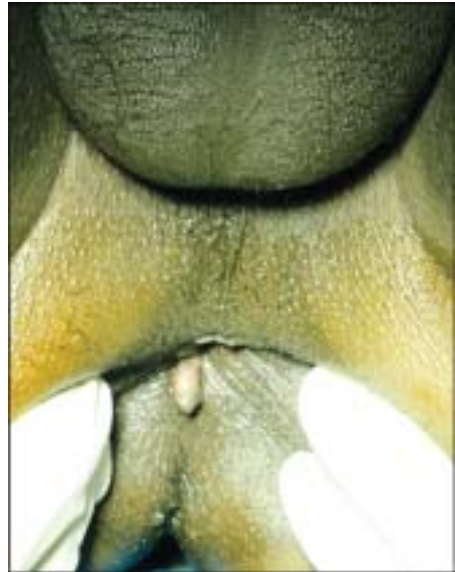


Fig. 22.50: Skin tag – external pile.

Fistula in Ano

It is a track lined by granulation tissue which connects perianal skin superficially to anal canal or rectum deeply. It usually occurs in a pre-existing anorectal abscess which burst spontaneously. *Other causes are:* Tuberculosis, carcinoma, Crohn's disease, ulcerative colitis, lymphogranuloma venereum, hydradenitis suppurativa.

Classifications

1. *Low level fistulas*—these open into the anal canal below the internal ring.
2. *High level fistulas*—these open into the anal canal at or above the internal ring.

Sentinel Pile ('sentinel' means guard)

It is commonly associated with *Fissure in ano* of chronic type wherein, in the lower part of fissure, skin enlarges and appears like guarding the fissure (**Fig. 22.50**).

Standard classification	Park's classification
Subcutaneous	Intersphincteric
Submucous	Transsphincteric
Low anal	Supralelevator
High anal	
Pelvirectal.	

Low-level fistulas: It presents with seropurulent discharge, along with skin irritation and one or more external opening may be present with induration of the surrounding skin. Often it may heal superficially but pus collects in the cavity forming an abscess which again discharges through same or new opening (**Fig. 22.51**). In case of ischiorectal fossa most often both fossae communicate with each other from behind causing *horse-shoe* fistula.



Fig. 22.51: Anterior fistula in ano with probe in place. Anterior low fistula has got straight track. Both internal and external openings are seen. Probing should be done in operation theatre under general anaesthesia. Probing should not be done in the ward as clinical method.

Goodsall's rule: Fistulas with an external opening in relation to the anterior half of the anus is of *direct type*. Fistulas with external openings in relation to posterior half of the anus, have a curved track may be of *horse-shoe* type, open in the midline posteriorly and may present with multiple external openings all connected to a single internal opening (**Fig. 22.52**). P/R examination shows indurated internal opening usually in the midline posteriorly. *Most of the fistulas*

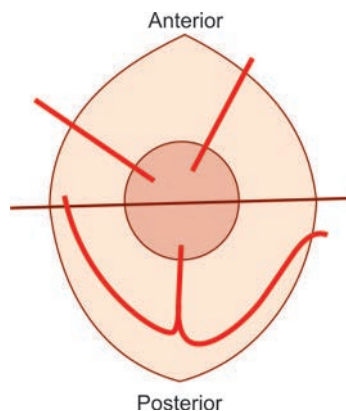


Fig. 22.52: Goodsall's rule. Anterior fistulas are having direct straight track. Posterior fistulas are having curved track with internal opening in the posterior midline.

are on posterior half of anus. Probing in the ward and fistulogram in the ward before surgery using Lipiodol is not advisable as it may cause recrudescence of inflammation.

Investigations: Chest X-ray, MR fistulogram (**Fig. 22.53**), ESR and Barium enema X-ray. If required, fistulogram is done only under anaesthesia. Tuberculous fistulas do not have induration, will have pale granulation tissue with watery discharge and they are most often multiple.



Fig. 22.53: MR fistulogram is very good (ideal) investigation of fistulas in ano.

High level fistulas: Its upper opening is at or above the anorectal ring. It is difficult to treat. Common causes are Crohn's disease; ulcerative colitis; trauma; carcinomas; foreign body. *Incontinence may follow after lay opening of these fistulas.*

Malignant Tumours of Anal Canal

Types: (1) *Squamous cell carcinoma* is the commonest type (**Fig. 22.54**). Predisposing causes: Papilloma, irradiation, dermatitis, long standing fistula in ano. (2) *Basaloid carcinoma*—It is rare, nonkeratinising squamous cell carcinoma. Highly malignant. (3) *Mucoepidermoid carcinoma*—arises near squamocolumnar junction. (4) *Basal cell carcinoma*. (5) *Melanoma*—blue/black in colour mistaken for thrombosed pile. (6) *Adenocarcinoma* from the anal glands in a pre-existing fistula in ano. *Squamous cell carcinoma* of anal canal usually present as a fungating or ulcerative growth, which spreads to inguinal lymph nodes.

Biopsy and FNAC of lymph nodes are the essential investigations.



Fig. 22.54: Anal canal carcinoma. Squamous cell carcinoma is commonest type – 80%.

Solitary Ulcer Syndrome

It is mainly thickening and disorganisation of muscularis mucosa with superficial ulceration. It is usually 4-12 cm from the anal verge in the anterior wall of the rectum. But often can occur in sigmoid colon. In 30% cases there are multiple ulcers. Often there will be inflammation and induration of the area without an ulcer. Condition is commonly associated with rectal prolapse. *Differential diagnoses* are carcinoma, tuberculosis, ulcerative colitis. *Avoid surgical excision in solitary ulcer syndrome.*

Proctitis

It is inflammation of rectal mucosa often with the inflammation of colon and anal canal. Types: Acute; Chronic.

Nonspecific—common

Ulcerative proctocolitis as part of ulcerative colitis.

Specific: Bacillary dysentery; Amoebic proctitis—common; Combined amoebic and bacillary; Gonococcal proctitis; Lymphogranuloma inguinale (LGV); Tuberculous proctitis; Bilharzial proctitis due to *Schistosoma haematobium*; Enema induced proctitis especially of herbal enemas. **Clinical features:** Pain per rectum and anum; tenesmus; passage of mucous and blood; frequently urge to pass stool; fever, loss of appetite; pain and tenderness in left lower abdomen; P/R is tender. **Investigations:** Sigmoidoscopy is more relevant than just proctoscopy; Stool study, stool culture; Mucosal biopsy; Serological tests; Relevant investigations like ESR, blood smear, and chest X-ray.

Proctalgia Fugax

It is sudden severe recurring pain in the rectum of unknown cause with segmental pubococcygeal spasm.

Features: It is common in young people may be due to stress, straining. Common at night, starts suddenly, lasts for few minutes and then subsides spontaneously. Pain is unbearable and severe with often constipation. Gradually subsides on its own. Occasionally only cutting of puborectalis muscle is required but with danger of developing incontinence.

Anal Incontinence

Continence of anal canal is maintained by two factors.

- 1) Normal rectal and colonic pressure and activity.
- 2) Normal pelvic floor function.

Types: *Urge incontinence*—Here rectal and colonic pressure and activity is increased but normal pelvic floor. *True incontinence*—Here rectal and colonic pressure and activity is normal but defective pelvic floor function. *Full incontinence*—Here rectal and colonic pressure and activity is reduced and also defective pelvic floor function. *Temporary*—Treated by reassurance. Often it is seen after Lord's dilatation. *Permanent*—Needs definitive therapy.

Causes: Irritable bowel syndrome, severe diarrhoea, prolapsed piles, rectal prolapse, old age, malnutrition, debilitating illness, congenital anomalies, trauma, surgeries, injury during childbirth in females, spina bifida, spinal tumours, spinal injuries and surgeries, malignancy, post-irradiation, psychological causes.

Evaluation of the patient: For specific causes; anorectal manometry; per rectal examination; sigmoidoscopy.

Anorectal Malformations (ARM)

It is imperfect fusion of the post-allantoic gut with the proctodaeum. Incidence is one in 4500 newborns.

Clinical Features

Newborn presents with inability to pass meconium, abdominal distension, features of intestinal obstruction,

improper anal dimple, sometimes with complaints of passing meconium per urethra. It can be associated with - Cardiac anomaly, tracheo-oesophageal fistula, renal anomalies and spinal anomaly.

Investigations

Invertogram: It is usually taken 6-12 hours after birth so as to allow air to reach the rectal pouch. Length between the rectal pouch and anal dimple marker is more than 2.5 cm in high anal fistula. In *low fistula*, rectal pouch is distal to the *Stephen's line (Pubococcygeal line)*. In *intermediate*, pouch is at the level of ischial spine (*Kelly's point*). In *high fistula* rectal pouch is proximal to the Stephen's line. **Murugassu's technique:** Through visible anal dimple, meconium is aspirated by passing a needle into the rectal pouch in sitting propped up position. Water soluble iodine dye is injected. Lateral X-ray is taken to study the level through Stephen line and Kelly's point.

Wingspread classification of ARM

('Wingspread'—name of the place where the conference was held)

Low	Intermediate	High
Covered anus Anovestibular fistula Anal stenosis Anal membrane It is below the level of pelvic floor (Puborectalis), Easy to diagnose and treat, good outcome Anoplasty; Anovestibuloplasty Anal dilatation, Incision of membrane	It occurs at the level of puborectalis with or without fistula.	It can be with or without a fistula into the bladder, urethra, uterus, vagina. Anorectal agenesis Rectal atresia Cloaca (only in females).

Examination in Urinary Diseases

Haematuria, retention of urine, increased frequency and burning urine are the common presentations of urinary diseases. It will cover wide range of diseases from inflammation to neoplasm from kidney to urethral end.

Haematuria

It is presence of red blood cells in urine. If it is more than 3/cu mm in uncentrifuged urine, it is pathological.

Types

- Gross (visible to unaided eye).
- Microscopic (>3 RBC's/HPF)

Early (initial) haematuria: Urethral origin, distal to external sphincter

Terminal haematuria: Bladder neck or prostate origin.

Diffuse (total) haematuria: Source in the bladder or upper urinary tract.

False haematuria: Discolouration of urine from pigments such as food colouring agents and myoglobin.

Silent haematuria is due to tumours of kidney or bladder unless proved otherwise.

Haematuria may be due to lesions of *urinary tract*; *adjacent organ diseases* causing transmitted inflammation to bladder or ureter (acute appendicitis, salpingitis, pelvic abscess) or malignant infiltration of ureter or bladder (carcinoma rectum or cervix or uterus or bowel infiltrating the bladder or ureter); *general diseases* like blood dyscrasias, scurvy, malaria, bacterial endocarditis, emboli, right heart failure, renal vein thrombosis; collagen diseases; *drug induced* like anticoagulants.

Isolated haematuria is due to bleeding from urethra to renal pelvis without any proteinuria, casts or cells.

Nephronal haematuria is due to blood entering the tubular fluid is trapped in a cylindrical mould of gelled Tamm-Horsfall protein to produce RBC casts containing degenerated RBC with clumps of haemoglobin. It suggests glomerulonephritis/tubulointerstitial injury, vasculitis. Haematuria with proteinuria carries bad prognosis.

Haemoglobinuria is presence of free haemoglobin in urine. It is due to intravascular haemolysis or strenuous exercise.

Causes (Fig. 23.1)

Renal injury
Urinary stones
Wilm's tumour
Tuberculosis
Renal cell carcinoma
Cystitis
Bladder tumour
Urinary bilharziasis
BPH, Carcinoma prostate
Renal infarct
Glomerulonephritis
Blood dyscrasias

Investigation

Urine culture and sensitivity:

Urine test for haematuria—*Benzidine test*; Ultrasound to look for the stone, tumour in the urinary tract; Cystourethroscopy to look for bladder or urethral pathology; IVU look for function of the kidneys; Urinary cytology for diagnosing urothelial malignancy; CT abdomen/pelvis depending on the location of the site of the cause; renal function tests; bleeding time, clotting time, prothrombin time, platelet count.

Retention of Urine

It is accumulation of urine in the urinary bladder. Kidneys excrete urine in normal quantity. But patient

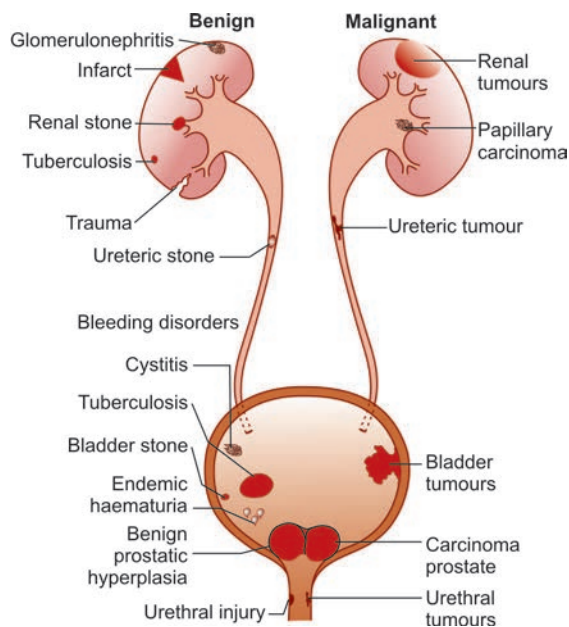


Fig. 23.1: Causes of haematuria.

passes only small amount of urine or does not pass urine at all causing *distended bladder*. In anuria due to renal failure, patient does not pass urine as kidney does not secrete any urine and bladder is not distended.

Causes

Urethral injury; benign prostatic hyperplasia (BPH); stricture urethra; carcinoma prostate; stone in the urethra; bladder tumour near bladder neck; phimosis with meatal stenosis; bladder neck hypertrophy; posterior urethral valve; prostatitis or urethritis; neurogenic causes like head and spinal injuries; post-operative cause; pelvic and perineal surgeries; drug induced retention of urine—anticholinergics, antihistamine drugs, antidepressants.

Clinical Features

Pain and swelling (fullness) in the suprapubic region; inability to pass urine; smooth, soft swelling that is tender in acute/non-tender in chronic, lying in hypogastric region which is dull on percussion; with increase desire to micturate on pressure in acute type but such desire will be absent in chronic type; upper border of bladder may be upto umbilicus; presence of features relevant to specific causes is seen in urethral meatus,

Causes of retention of urine

Urinary causes

Phimosis, meatal stenosis
Stricture urethra, rupture urethra, posterior urethral valves, urethritis, stone in urethra, urethral tumours
BPH, carcinoma of prostate, prostatic abscess, prostatitis
Bladder stone, tumour, bladder neck contracture (Marion's disease)

Compression from outside

Carcinoma cervix, ovarian tumours, rectal and pelvic tumours, paraphimosis, pregnancy

Neurogenic causes

Spinal injuries
Spinal diseases – Pott's disease
Spinal cord diseases – disseminated sclerosis, tabes dorsalis, transverse myelitis

Other causes

Postoperative (perineal surgeries); tetanus, hysteria, drugs like anti cholinergics, muscle relaxants, tranquilizers

phimosis, bulb of urethra, prostate, nervous diseases. *Digital examination of the rectum* feels backwards and downward displacement of the prostate by distended bladder which feels cystic. Neurological examination is essential. *Cricket ball bladder* in infants is due to retention of urine due to posterior urethral valve.

Acute retention: It is rare. It is sudden inability to pass urine. It is painful distension of the bladder. It is seen in urethral trauma, due to anaesthesia, or surgery (perineal/abdominal). There is increased desire to pass urine.

Chronic retention is gradual collection of urine in the bladder due to ineffective emptying of the bladder completely. Bladder is distended and is painless. It is common in elderly. Frequency, difficulty in urination, overflow incontinence is common. Infection in such chronic retention makes it painful.

Acute on chronic retention: Patient is having chronic obstructive condition like BPH; due to infection, acute inflammation and oedema of mucosa of urethra sudden total blockage sets in causing acute on chronic retention of urine.

Investigations

US abdomen; Blood urea, serum creatinine; Urine microscopy.

Note: In retention of urine prostate should not be assessed during rectal examination. It should be assessed only after catheterization and emptying the bladder.

Increased Urinary Frequency

Normal urinary frequency is 5-6 times a day. Increased frequency may be observed if fluid intake is more than normal; if there is increased urine formation like in diabetes mellitus or interstitial nephritis; if total quantity is normal but frequency increases due to some pathology in the urinary system. Diurnal frequency is observed in vesical stone which causes irritation of trigone of bladder during day time. Night frequency is common in BPH. Frequency is equal during day or night time in cystitis. Increased frequency, acid urine, sterile pyuria are features of renal tuberculosis. Gonococcal urethritis also causes increased frequency.

Causes of increased frequency

In the kidney—Stone; tuberculosis, pyelitis.

In the ureter—Stone, ureteritis.

In the urinary bladder—Stone, cystitis, pelvic infections like salpingitis, appendicitis, compression by ovarian cyst, fibroid, retroverted uterus, malignant infiltration of bladder by carcinoma rectum/uterus.

In the prostate—Prostatitis, BPH, carcinoma.

In the urethra—Stone, gonococcal urethritis, stricture, balanitis.

Definitions of Various Terms

Polyuria is urine volume above 3 litre/day. It is seen in diabetes insipidus, diabetes mellitus, diuretic phase of acute tubular necrosis, on diuretic drugs, hypercalcaemia, hypokalaemia, polydipsia (increased thirst), and salt losing nephritis.

Nocturia means volume of urine passed at night becomes equal or exceeds of day time. It is seen in BPH, diabetes mellitus, diabetes insipidus, cardiac failure on drugs, insomnia.

Dysuria is pain/burning during urination.

Strangury is painful desire to urinate which starts in bladder radiating down into the urethra; but pain will not be relieved; urine also will not be passed.

Frequency of urine refers to passing urine at frequent intervals even though bladder is not full but patient feels sense of fullness of bladder due to irritable bladder.

Urgency is an exaggerated sensation to urinate.

Incontinence is inability to retain the urine in the bladder.

Enuresis is involuntary passage of urine during sleep or at night. It is normal in children below 2 years of age.

Pyuria means presence of pus cells in urine > 10 WBCs/cu mm of uncentrifuged midstream urine in females; > 3/cu mm in males. Contamination by vaginal secretions is more in females. Pus cells > 5/high power field in centrifuged urine in either male or female is also called as pyuria.

Sterile pyuria is presence of WBCs but culture is sterile – seen in treated urinary infections by antibiotics or in urinary tuberculosis/pregnancy/cyclophosphamide chemotherapy/prostatitis.

Proteinuria: Normal adult excrete protein upto 150 mg/day of which 5-15 mg is albumin and remaining are others about 30 types proteins and Tamm-Horsfall mucoprotein (renal cell glycoprotein). Excretion of protein more than 150 mg/day is called as *proteinuria*. 150 mg –1 gram/day is called as mild proteinuria; 1-3.5 gram/day is moderate; > 3.5 gram/day is massive.

Microalbuminuria: It is early morning urine albumin/creatinine ratio > 3 or albumin excretion rate of 20-200 µg/minute or 30-300 mg/24 hours. Excretion of mainly albumin suggests glomerular disease—*selective proteinuria*.

Orthostatic proteinuria is proteinuria seen in daytime collected urine which is < 1 gram/day with absence of protein in urine collected at early morning. It is seen in 2-5% of adolescents.

Pneumaturia is passing air bubbles in the urine. It suggests vesicocolic/vesicointestinal fistula. Often it may be seen in emphysematous pyelonephritis due to gas producing organisms (in diabetics).

Azotaemia is an increase in blood urea and serum creatinine.

Oliguria means decreased urine output which is inadequate to maintain life – less than 400 ml/day.

Anuria means absence of urine flow. It is usually referred to absence of excretion of urine in renal tubules. It is referred as less than 50 ml/day. *Anuria* due to obstructive uropathy is mechanical block.

History

Age

Carcinoma of prostate is a disease of elderly after 60 years. Benign prostatic hyperplasia is common after 50 years. Polycystic kidney disease is common in middle aged even though it is congenital. Renal calculi are common after 30 years. Renal cell carcinoma is common in middle aged. Wilm's tumour is seen before the age of 4.

Sex

Stone disease and renal cell carcinoma are common in males. Cystitis is common in females.

Residence

Bladder stone is common in Punjab and Rajasthan. Schistosomiasis is common in Africa, Iraq, and Iran.

Occupation

Bladder carcinoma is common in industrial workers. Stone disease is common in manual labourers who dehydrate commonly.

History of Present Illness

Pain

Renal pain: It is dull ache in the renal angle between erector spinae and 12th rib. It is constant pain in the loin often in the upper outer quadrant of abdomen spreading along subcostal area towards umbilicus. Patient places his thumb in front pointing towards umbilicus and fingers over renal angle. It is due to stretching of renal capsule and renal pelvis. Causes of pain may be stone, infection, tuberculosis, haemorrhage in the cyst or tumour. Often opposite

kidney may undergo hypertrophy and cause pain in opposite loin (renorenal reflux). When renal pain is severe, this severity may vary from time to time mimicking renal colic. But such renal colic, often named as, is a misnomer as it is never a gripping type of pain and it persists here in between severity of episodes. Onset, progression, severity should be asked for.

Ureteric pain: It is typical. It is due to spasm of the muscular tube of ureter and stretching of the capsule of pelvis and ureter. It originates in the loin in renal angle radiates along the course of the ureter along the waist, towards the groin to reach the penis and scrotum in males, and labia majora in females. It also radiates to upper part of thigh through genitofemoral nerve (L_1). This is because upper ureter and testis has got common innervation (T_{11} , L_{12}) and lower ureter and upper thigh have got common innervation (L_1). This *colicky pain* is gripping in nature, becomes very severe and subsides completely with a pain free interval, and appears again in a waxing and waning manner. Jolting movements will precipitate the pain. Pain is usually associated with nausea and vomiting due to reflex pylorospasm. Stone in the upper ureter causes pain in the loin radiating to the testis; pain in the lower ureter radiates to McBurney's point in right side mimicking acute appendicitis, to amebic point in left side mimicking amebic colitis/diverticulitis (T_{12} - L_1). Stone and clot are the common causes of ureteric obstruction (**Fig. 23.2**).

Vesical pain: It is often suprapubic, midline dull ache type. Occasionally it may be severe pain also. Causes are—acute retention of urine, bladder stone, tuberculosis, schistosomiasis of bladder. In vesical calculus, on standing stone comes in contact with trigone of the bladder which is very sensitive and causes pain whereas it is less painful on lying down as stone moves/floats towards fundus. Pain originates in suprapubic region and gets referred to tip of the penis. Children will scream suddenly by holding and pulling the prepuce. Haematuria at the end of urination is common. Chronic retention of urine hardly produces any suprapubic pain unless it is infected. Cystitis often causes referred pain in distal urethra. Strangury is common.

Prostatic pain: It is peculiar aching discomfort or fullness in the perineum and rectal area. Referred



Fig. 23.2: Patient hold the loin with thumb pointing towards umbilicus; fingers behind.

lumbosacral pain may be the occasional symptom in prostatitis. Prostatic abscess causes throbbing pain. BPH or carcinoma prostate usually will not cause any pain. Carcinoma prostate when once becomes locally advanced can cause pain. Back pain in carcinoma prostate is probably due to secondaries in sacrum (osteosclerotic).

Urethral pain: It is scalding pain occurring at the end of the micturition usually due to urethritis.

Swelling

Duration of swelling, mode of onset, progress one side or both sides should be asked like in any swelling. Painless swelling in the loin often with haematuria could be due to renal cell carcinoma. Painless swelling in a child below 5 years is probably due to Wilm's tumour. Swelling appearing and disappearing often with pain is probably due to *Dietl's crisis*—*intermittent hydronephrosis*. Bilateral renal swelling could be as a result of bilateral hydronephrosis due to congenital PUJ obstruction or polycystic kidney disease. One kidney may be involved early than the other.

Haematuria

Blood in the urine is an important history in the urinary system. Patient often first approaches doctor for

haematuria. It may be due to many causes – stone, tumour, infection. Quantity and relation to micturition is important. *Early haematuria*, i.e. at the beginning of the urination is due to urethral cause; *terminal haematuria*, i.e. at the end is due to bladder diseases; *total haematuria* is throughout the urination and is probably due to renal or prerenal causes. Association of pain and type of pain should be asked. Red urine (without red cells) is probably due to diet (Beet root), phenolphthalein, cakes, fruit juices (rhodamine B). Haemolytic diseases can cause haemoglobinuria with red urine.

Retention of Urine

Acute retention is painful; chronic is painless. BPH, carcinoma of prostate, urethral causes to be considered. It should be differentiated from anuria.

Change in the Stream of Urine

Normal force and stream in urination is projectile. It becomes vertical and slow in old age due to BPH. Straining improves the stream in urethral stricture. In enlargement of median lobe of prostate straining reduces the stream as median lobe comes closer to internal meatus to block it. Bladder stone or pedunculated bladder papilloma may cause sudden stoppage of stream by blocking the internal opening of the bladder. Patient can pass urine by change of posture.

Frequency of Micturition

Increased frequency of urination is observed in cystitis and BPH. There will be retention of urine or increased residual urine in these patients. Night frequency is typical feature of BPH.

Anuria or Oliguria

In anuria bladder is empty as there is no urine formation. It is a feature of renal failure. It may be pre-renal; renal or post-renal.

History of Incontinence

True—patient passes urine without warning and bladder is empty. *False*—it is overflow incontinence in a distended full bladder. *Urge*—when there is

urgency to pass urine, few drops of urine will be passed; seen in cystitis in women, BPH in men. *Stress* – urine comes out while straining like laughing, coughing due to weak sphincter.

Urethral Discharge

It is early morning gleet (glairy fluid) in prostatitis. Profuse and purulent in gonococcal urethritis.

History of hiccough, oedema feet, thirst, vomiting, insomnia are other history to be noted.

Other Symptoms

Fever, chills, rigors are features of acute urinary infection. If associated with backache, abdominal pain and distension then it could be due to acute pyelonephritis.

Colonic, liver or pancreatic disease like features can develop due to close proximity and irritation.

Past History

History of tuberculosis, drug intake, treatment earlier for renal failure/stone diseases/obstructive uropathy/surgeries should be asked.

Family History

Many urinary diseases may run in family. Chronic renal failure/polycystic disease of kidney may be familial.

General Examination

Anaemia may be a feature of chronic renal failure. Dry tongue is a feature of renal failure. Altered respiration, oedema feet and face, often anasarca are seen in renal failure. Hypertension is often seen in polycystic kidney disease, renal cell carcinoma, renal artery stenosis, and hydronephrosis.

Local Examination

Kidney

Inspection

Kidney mass is inspected well in *sitting position*. It may not be obvious in lying down position. *Fullness* in the loin is seen from behind (**Fig. 23.3**). *Renal angle* (between lateral border of sacrospinalis and 12th rib)



Fig. 23.3: Loin should be inspected from behind in sitting posture.

is inspected. In perinephric abscess *scoliosis* of lumbar spine with concavity towards affected side is typical. Here bending the trunk away from the side of the lesion causes pain. Redness, oedema over the region suggests inflammation and abscess. In bilateral diseases both loins are full. Inspection is also done from front.

Palpation

Normal kidney is *not* palpable usually. Mobile kidney may be palpable if it is having mild hydronephrosis due to recurrent kinking. *Right side* kidney is palpated from *right side*; *left side* kidney is palpated from *left side*. Kidney is *bimanually palpable*. In enlarged *right* kidney—left hand fingers are placed behind loin over renal angle to lift the kidney; right hand is placed in front below the costal margin over abdomen; patient is asked to take deep breath; during every phase of expiration when muscle is getting relaxed right hand in front is pushed posteriorly to feel the kidney. Its size, shape, surface, consistency, extent should be assessed. Movement with respiration is confirmed during deep inspiration. Usually enlarged kidney moves downwards during inspiration. In perinephric collection, pyonephrosis with inflammation around and locally advanced renal cell carcinoma kidney may



Figs 23.4A and B: Bimanual palpation of the kidney.

not move with respiration. Hydronephrosis and cystic diseases show cystic swelling. Carcinoma, tuberculosis causes solid, hard irregular swelling. Renal mass also can be felt in *side position* (towards normal side). In newborn, kidney mass can be palpated well with fingers of the hand in renal angle and thumb of the same hand in front. Both sides are palpated together with both hands (**Figs 23.4A and B**).

Ballottement/ballottability: One hand is kept behind in the loin; other hand is kept in front over the abdomen. When sharp short forward pushes are made by fingers behind to displace the kidney mass front (displacing hand); bouncing impact is felt by the fingers in front (feeling/watching hand). Very large mass (large hydronephrosis), locally advanced renal cell carcinoma, mass with inflammation surround will not be ballottable.



Fig. 23.5: Murphy's kidney punch.

Murphy's kidney punch: In sitting position, patient folds the hands in front to stretch the back to elicit the sign better, and using the thumb examiner presses at renal angle to elicit tenderness (**Fig. 23.5**).

Pitting oedema may be evident at renal angle in perinephric abscess.

Features of renal mass

- Reniform shape
- Mass in loin
- Moves with respiration downwards
- Bimanually palpable
- Ballottable
- Band of colonic resonance in front
- Dullness in renal angle (normally renal angle is resonant due to colon)
- Always can insinuate between costal margin and mass

Differential diagnoses for renal mass

- Splenic mass
- Colonic mass
- Adrenal mass
- Liver mass in right side

Percussion

Renal mass is resonant *in front* due to colonic band. Renal angle is percussed in *sitting posture*. Both sides should be percussed to compare. *Normal* renal angle is *resonant* due to colon. Inflation of air by passing rubber catheter per rectally makes (normal) resonance



Fig. 23.6: Percussion over renal angle in sitting position. Renal angle is normally resonant.

of normal renal angle better on percussion (*Baldwin's method*). When kidney *enlarges* it displaces the colon in front and medially making renal angle *dull on percussion* (**Fig. 23.6**).

Auscultation Around Umbilicus (Right or Left)

It is done to hear systolic bruit in renal artery aneurysm or renal artery stenosis.

Ureter

Usually cannot be felt. Distal part may be felt per vaginally or per rectally occasionally in ureteric stone or tuberculosis.

Bladder

Empty bladder lies in the pelvis; once it fills with more than 150 ml of urine it is palpable in hypogastric region, suprapubically. Fully distended bladder will reach up to umbilicus. Fullness is *visible* in suprapubic region up to umbilicus on inspection. *Soft/elastic* tender (in acute retention) mass is felt on palpation. It is *dull* on percussion. Distended bladder can be felt on *rectal* examination. *Cricket ball* like hard bladder is typical of posterior urethral valve in children. Bimanual palpation of bladder is done under general anaesthesia with index finger of one hand (right) in rectum pushing the bladder forward and other hand placed in suprapubic region to feel the bladder. It is useful in



Fig. 23.7: Extrophy of bladder showing posterior wall of urinary bladder.



Fig. 23.8: Epispadias.

bladder tumours. Base of bladder is normally not felt. Stone in this area and tumour can be felt especially with bimanual palpation under general anaesthesia (**Figs 23.7 and 23.8**).

Prostate

It is palpated by digital examination of rectum in left lateral or knee-elbow position. Lithotomy position is used to palpate prostate bimanually under general anaesthesia. Size, surface (smooth in BPH, nodular or irregular in carcinoma), consistency (normal is rubbery; in BPH it is firm; in carcinoma it is stony hard), median groove (it is obliterated in carcinoma), mobility of rectal mucosa, tenderness (tender is

prostatitis or prostatic abscess) is looked for. Usually median lobe is not felt per rectally but after passing a urethral bougie median lobe can be felt. Prostatic massage is important in chronic prostatitis which often has vague symptoms.

Seminal Vesicles

Knee elbow or Picker position is used to palpate seminal vesicles. Normally they are not felt. It is felt in infection as cystic tender mass; in tuberculosis as firm irregular mass.

Urethra

Discharge, fistula, tenderness are looked for; bulbar urethra is felt by lifting the scrotum and feeling in midline (in stricture urethra thickening and crescent like feel will be present). Stone may be felt. Often stone may be visible near the tip of urethra (**Figs 23.9 and 23.10**).



Fig. 23.9: Peri-urethral abscess.



Fig. 23.10: Phimosis may be cause for obstructive uropathy.

Systemic Examination

Lungs should be examined for secondaries from renal cell carcinoma through inferior vena cava (blood spread, lung is the commonest site of secondary in RCC). Parathyroid enlargement may occur in hyperparathyroidism with multiple stones in the kidney.

Examination of Urine

24 hour urine sample is checked. In renal tuberculosis early morning urine sample is needed for AFB analysis. Colour, clearness/cloudiness, quantity should be checked. Midstream urine is collected for bacterial analysis and culture. Midstream urine shows sediments if there is phosphate or pus. If after adding acetic acid sediment disappears, it is due to phosphate. Bile in the urine gives greenish brown colour with yellow brown foam on shaking. It becomes greenish after sometime due to oxidation of bilirubin to biliverdin. In patient with porphyria urine is orange coloured and on exposure for few hours to air, upper part of the collected urine becomes amber coloured. Cascara, senna, rhubarb turns urine brown; salicylic acid, pyridium turns to reddish yellow; sulphonals turns to pink; methylene blue to greenish in small amount blue in large quantity. Beeturia is due to betacyanin pigment in urine after eating beetroot. Urine will be milky white in chyluria.

Two glass urine test or *three glass urine test* is often significant. First sample is from urethra and prostate, second sample is from bladder, third from kidneys. Prostatic threads may be present in first sample suggestive of chronic prostatitis. Urine should be analyzed for crystals, bacteria, pus cells, malignant cells. Sterile acid pyuria is a feature of tuberculosis. Such urine should be assessed for AFB, culture. Normal pH of urine is 4.5-7.5. In diabetic acidosis it is below 4.5; above 7.5 in presence of urea splitting organisms. After 12 hours of overnight fluid restriction urine is collected for specific gravity. Early morning sample is used. Normal is 1.020. If it is less than 1.010 it suggests renal failure.

Selective urine sample from each kidney for specific analysis can be taken by ureteric catheterization using cystoscopy.

Blood Evaluation

Polycythaemia may be a feature in renal cell carcinoma. Anaemia is seen in renal failure. Tumour markers like CEA in RCC or AFP in bladder tumour may be raised.

Renal Function Tests

(1) Proteinuria. (2) Specific gravity. (3) PSP test – after passing urine phenolsulphonaphthalein is injected intravenously; patient is given 20 ml of water every half hourly; urine is collected every half hourly and assessed for PSP dye. If 50-60% of dye is present in first half an hour urine sample; 15% in next half an hour sample, it suggests adequate renal blood flow and tubular function. (4) Creatinine clearance value normally is 70-140 ml/hour. It suggests glomerular filtration rate. Chromium 51 labeled EDTA is more accurate method. (5) Blood urea is 20-40 mg/100 ml; serum creatinine is 1.4 mg/100 ml.

Plain X-ray-KUB (Kidney, Ureter Bladder)

Preparation of the patient: Enema/bowel wash/laxative is given on the previous day and the patient is asked to fast in order to reduce the bowel gas shadows in X-ray. High penetration X-ray is taken in supine position which covers from *pubic symphysis to lower two ribs*. Often films are taken in deep inspiration, after full expiration and also in standing positions.

Interpretation of the film: First bony parts are looked for, i.e. the hip, pelvis, lumbar vertebrae for fractures, scoliosis, spina bifida, secondaries in the spine. **Kidney shadow**—kidney shadow is visualized in plain X-ray KUB due to difference in the density between kidney (high vascularity) and perinephric fat (low vascularity). Findings that are looked for are size, location, calcification and stones. In congenital absence or ectopic kidney this shadow may be absent. It may be enlarged in hydronephrosis, renal cell carcinoma. It extends normally from upper level of 1st lumbar vertebra to lower level of 3rd or middle of 4th lumbar vertebra. Right kidney shadow is lower than left due to liver in right side. In children perinephric fat is absent and so kidney shadows are not visualised. Renal stones are radio-opaque commonly (90%) and so are visualized in plain X-ray KUB. Renal stone changes

its position in films taken during inspiration and expiration. In lateral view renal stone overlaps/superimposes the vertebrae but gallstones are located in front of the vertebrae. **Psoas shadow**—It is visualized well in normal KUB. *It is obliterated* in enlarged kidney; scoliosis due to inflammatory or infiltrative causes; malignancy; tuberculous spine with cold abscess (psoas abscess); splenic injury (left sided shadow); retroperitoneal tumours. **Ureteric line** is looked for any radio-opaque oval shadow (ureteric stone). It runs along the tips of the *transverse processes of the lumbar vertebrae*, crosses the *sacroiliac joints* and heads upto a point medial to the *ischial spine*. Bladder, prostate and urethral areas are visualised for any lesion.

Differential diagnosis for radio-opaque shadow which mimics renal stone

- Calcified lumbar or mesenteric lymph node.
- Gallstone. (10% are radioopaque).
- Concretion in appendix.
- Phleboliths.
- Ossified tip of 12th rib.
- Chip fracture of transverse process of vertebra.
- Calcified renal tuberculosis.
- Calcified suprarenal gland.
- Drugs or foreign body in the alimentary canal.

Intravenous Urogram (IVU)

Procedure

Renal function must be normal. Overnight fasting for 8 hours is advised. Laxatives are given to reduce bowel shadow and get a good quality film. First a plain X-ray KUB is taken (IVU should not be read without doing KUB). Then 1 ml test dose of **Sodium diatrizoate (Urograffin)** or **Meglumine iothalamate IV** is injected and waited for 5-10 minutes. If no adverse reaction occurs, then full dose—1 ml/Kg body weight of urograffin is given intravenously (about 40-50 ml). X-ray KUB is taken in 3-5 minutes which will show the nephrographic and secretory function of the kidneys. Later after 10, 15 minute and then 20-30 minute films are taken. Further films are taken depending on the need. Films are taken earlier in children as excretion is quicker. Film can be taken as late as 72 hours. Late films show bladder pathology as well as residual urine.

In case of *renal failure* with high blood urea, dose of dye is increased to 2 ml/Kg body weight to get

Indications	Findings
1. Hydronephrosis	Clubbing of calyces
2. Congenital anomaly a. Horse shoe kidney b. Duplex kidney and Double ureter c. Ureterocele d. Polycystic kidney disease e. Retrocaval ureter	Flower vase appearance Adder (cobra) head appearance Spider leg appearance Reverse 'J' sign with hydronephrosis
3. Renal cell carcinoma	Irregular filling defect
4. To see the function of the kidneys in bilateral diseases	Bilateral stones, obstructive uropathy
5. After surgery for urinary diseases.	To see the function of kidneys and outcome of the surgery
6. Renal injury	To see the function of other kidney (A very specific investigation)
Contraindications: 1. Iodine sensitivity- may go for anaphylaxis. Hence all precautions must be taken and essential drugs should be available while doing IVU. 2. Multiple myeloma and hypergammaglobulinaemias (Acute renal failure may be precipitated due to dehydration and also dye makes an insoluble complex with Bence-Jones proteins which block the renal tubules). 3. Toxic thyroid.	



Fig. 23.11: Bilateral hydronephrosis with hydroureter—IVU film.

a better film -**Infusion IVU**. Often diuretics are used in these patients to have better secretion. Lower abdominal compression can be done to have better definition of calyces but not done in children and patients with abdominal aortic aneurysm. **Minute IVU**- In case of renal artery stenosis, within first minute many films are taken to get a nephrographic shadow (where a small, concentrated kidney is seen). **Non visualization of kidney:** No contrast is seen in the film even after 12 hours (**Fig. 23.11**).

Retrograde Pyelography (RGP)

Indications: Failure of showing any secretions in an IVU as late as 72 hours film; Urinary tuberculosis; Urothelial tumours from the renal pelvis.

Procedure: Under G/A cystoscope is passed. Ureteric orifice is visualized. Ureteric catheter is passed. 3-5 ml of dye, sodium diatrizoate is injected. Patient is put in 15° head down position to allow the dye to reach upper urinary system. X-ray is taken. Often 4-6 ml of air is infused afterwards to differentiate stone in pelvis from papillary tumour—*pneumopyelography* (**Fig. 23.12**).

Advantages: Prior to dye injection selective urine sample can be taken from each ureter. Brush biopsy

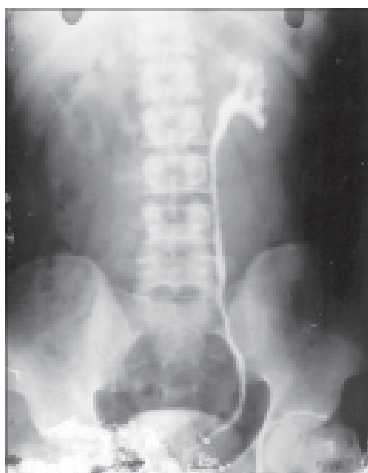


Fig. 23.12: Left RGP-normal study.

from suspected urothelial tumours of upper urinary tract can be taken; Better-delineation of anatomy is possible (due to more concentration of dye).

Disadvantages: Anaesthesia is required and is laborious.

Antegrade Pyelogram/Urogram

Under fluoroscopy or US guidance 18 gauged 15 cm length needle is passed into the dilated renal calyx and dye is injected to delineate the pelvicalyceal system.

Renal Angiogram

Indications: Renal artery stenosis; renal artery atheroma; renal artery aneurysm; occasionally renal cell carcinoma; arterial anomalies.

Procedure: Retrograde Seldinger technique: Through femoral artery, selective angiogram is done to visualise tumour vascularity, narrowing, anomalies. 10 exposures in 10 seconds are taken. Therapeutic embolisation, transluminal balloon angioplasty for renal artery stenosis can also be done. Translumbal approach for angiogram (through aortogram) is also used.

Complications: Paraplegia; embolism; dissecting aneurysm; bleeding; renal tubular necrosis.

Renal pharmacangiogram: Noradrenaline is injected along with the dye. Normal vessels will constrict in

response to noradrenaline. But since tumour is autonomous, vessels in renal cell carcinoma do not respond to noradrenaline and so *tumour blush* is seen. Digital subtraction angiography (DSA) is very useful to assess the renal tumours.

Flush Venogram

It is very useful to assess IVC and renal veins in RCC.

Micturating Cystourethrography (MCU)

Indications: Vesicoureteric reflux; Posterior urethral valve.

Procedure: Catheter is passed into the bladder. Dilute iodine dye is infused. X-ray is taken during micturition. *Free reflux* is looked for. X-ray is taken on applying pressure over the suprapubic region. *Pressure reflux* is studied. **Vesicoureteric reflux** is graded depending on the severity of the reflux: *Grade I*—ureters are seen; *II*—ureters and pelvis are seen; *III*—ureters, pelvis, calyces are seen; *IV*—with grossly distended calyces; *V*—tortuous elongated serpentine ureters. It can be unilateral or bilateral. Often it is associated with posterior urethral valve. It is often complicated by infection, pyonephrosis and renal failure (**Figs 23.13 and 23.14**).



Fig. 23.13: Micturating cystourethrogram showing concomitant existence of posterior urethral valve (causing dilatation of proximal urethra) and vesicoureteric reflux (both sides).



Fig. 23.14: MCU showing bladder diverticula.

Ascending Urethrogram

It is the investigation of choice for *stricture urethra*. Red rubber catheter is passed into the external meatus. Water soluble iodine dye is injected through the catheter. Oblique X-ray films are taken to visualize the urethra. Site, size, extent of stricture and extravasation can be found out in urethrogram. It is useful tool to see diverticulum, dilated prostatic ducts, bladder neck obstruction.

Isotope Renography

A measure of individual kidney function is obtained by this method using a gamma camera. Radio labelled Technetium 99m **DMSA** (Dimercapto succinic acid) **or DTPA** (Diethylene Triamine Penta acetic acid) is given intravenously. It shows: (a) *Early* vascular phase; (b) *Then* secretory phase; (c) *Later* excretory phase. This allows the assessment of renal plasma flow to each kidney and the efficiency and effectiveness of pelvi calyceal excretion also.

Problems: (1) Positioning of counters. (2) Often difficult to differentiate from muscle mass. It is only a supportive investigation.

Cystoscopy

To examine urethra, bladder, ureteric orifice—for any pathology (tumour, infection); To visualize any bladder fistulas; To treat—Urethrotomy (in stricture urethra), TURP for BPH and carcinoma prostate, bladder tumour resection, bladder stone removal

(cystolithotripsy, cystolitholapaxy), ureteric catheterisation, fulguration of posterior urethral valve.

Contraindication: Acute cystitis and prostatitis.

Types: Rigid; Flexible. Procedure: Patient is placed in lithotomy position. Under G/A or spinal anaesthesia after cleaning and draping, cystoscope is passed using continuous *glycine irrigation* (to avoid TURP syndrome). The parts of the urethra are visualized while passing the cystoscope-urethroscope. Once bladder is reached, it is looked for diverticula, hypertrophy and other pathologies. Ureteric orifices are visualised at 4 and 8 o'clock positions. Normal urinary efflux is noted. If 5 ml of sterile indigocarmine is injected intravenously, a blue jet of dye coming down from the ureteric orifices can be seen in 3-5 minutes—chromocystoscopy. In haematuria of suspected renal cause, cystoscopy is done during active bleed; but if suspected bleed is from bladder cystoscopy is repeated once bleeding stops (**Fig. 23.15**).

Complications: Urethral injury, bleeding, water intoxication.



Fig. 23.15: Cystoscopy is being done (Courtesy by Dr Ashok Pandith MCh urologist Mangalore).

Urethroscopy

Anterior urethroscopy is done in stricture, chronic urethritis, and foreign body using air inflation. Posterior urethroscopy is done with irrigation fluid to see prostatic urethra with verumontanum, sinus pocularis, ejaculatory ducts, prostatic ducts (**Fig. 23.16**).



Fig. 23.16: Cystoscopy/urethroscopy showing urethral stricture.

CT Scan

It is used to assess tumours, lymph node status, IVC spread, local infiltration, metastases in liver, lungs, etc. It is very useful in inflammatory condition also. CT scan is superior to MRI. PET scan is better in staging urological tumours (**Fig. 23.17**).

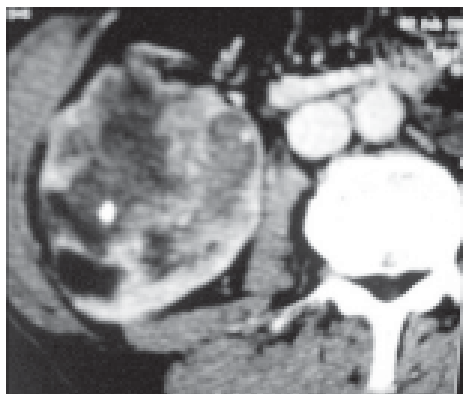


Fig. 23.17: CT scan showing features of renal cell carcinoma.

Horse-Shoe Kidney

It is a developmental anomaly where there is failure of complete ascent of kidneys with the fusion of lower or upper poles. This condition is common in males. Fusion of lower pole is common (rarely upper poles) (**Fig. 23.18**).



Fig. 23.18: Horse-shoe kidney.

Commonest site is in front of 4th lumbar vertebrae. The part in front of the vertebra is called as *isthmus*. It has blood supply which freely communicates one kidney to other. Isthmus usually lies in front of aorta.

Clinical Features

Presents as a fixed, nonmobile, firm mass in the midline at the level of 4th lumbar vertebra which is resonant on percussion. It is more prone for infection, stone formation, hydronephrosis, and tuberculosis.

Diagnosis

IUV—Medialisation of lower calyces and curving of ureter like a *flower vase*.

Polycystic Kidney Disease (PCKD)

Adult PCKD is inherited as autosomal dominant disease. It is bilateral and presents in third decade. One side presents little earlier than other side. **Other associations:** Polycystic diseases of liver (18%), pancreas and lungs. Berry's aneurysm in the circle of Willis. Cyst formation occurs at the *junction of the distal tubule and the collecting duct*. Grossly it contains multiple cysts with a clear or brownish fluid (due to haemorrhage).

Clinical Features

Bilateral palpable renal mass; loin pain; haematuria; infection; hypertension; uraemia. renal mass which is lobular, firm, mobile, moves with respiration, ballottable, with dull renal angle and resonant band in front. Pain is due to stretch of renal capsule or haemorrhage into a cyst. Haematuria (25%) is due to overdistended cyst rupturing into the renal pelvis. Infection is due to stasis. Hypertension (75%) is

common. Uraemia occurs in late stage due to renal failure.

Differential Diagnosis

Renal cell carcinoma; hydronephrosis; solitary renal cyst.

Investigations

US; IVU—*Spider leg pattern* (smooth, regular) with an elongated compressed renal pelvis, narrowed and stretched calyces; Blood urea and serum creatinine; Urine shows low specific gravity below 1.010; traces of albumin without casts or cells. Infection is common.

Solitary Renal Cyst

Solitary renal cyst is never congenital. It is due to an earlier trauma or infection resulting in blockage of tubule leading to cyst formation. It is usually unilateral, presents as a renal mass which is smooth, often tender if infected or haemorrhagic.

Duplication of Renal Pelvis

It is the most common congenital anomaly of the upper urinary tract (4%). It is usually unilateral; common in the left side. In 3% of cases it is associated with duplication of ureter. Upper renal pelvis is small, drains the upper calyces. Lower renal pelvis is larger, drains from middle and lower calyces. Double ureter when associated, may be *partial* where two ureters join in lower third or *complete* where upper ureter opens into the bladder at a lower level and lower ureter opens into the bladder at the upper normal ureteric orifice. This is called as *Weigert Meyer Law*. In partial duplex, there is reno-renal reflux resulting in infection, stone formation and hydronephrosis.

Investigation: IVU—diagnostic. US to look for complications. Cystoscopy will show **double ureteric orifices** on the same side.

Retrocaval Ureter

It is due to developmental defect of IVC, as a result of which ureter passes behind the IVC, causing right sided hydronephrosis with upper third hydroureter. IVU shows hydronephrosis with *reverse J sign*.

Ureterocele

It is a *cystic enlargement of the intramural portion of ureter* due to congenital atresia of the ureteric orifice. Its wall contains mucous membrane only. It is common in females, often it is bilateral (10%). It causes hydronephrosis, infection, and calculi formation.

Stephen classification: Stenotic, sphincteric, sphincterostenotic.

Investigation: IVU—shows *Adder-head* appearance or *cobra head* appearance. Cystoscopy shows translucent cyst which is thin walled surrounding the ureteric orifice.

Perinephric Abscess

Causes

Infection of a perinephric haematoma; Perforation through renal capsule from pyonephrosis or renal carbuncle; Tuberculous perinephric abscess; Extension of cortical abscess; Haematogenous spread; Extension of appendicular abscess; Periureteral lymphatic spread.

Clinical Features

High fever; fullness and rigidity; tenderness in the loin; scoliosis with concavity towards the abscess.

Investigations

Total count is increased. *Plain X-ray KUB*—Obliteration of psoas shadow, scoliosis, elevation of hemidiaphragm.

IVU—Two films are taken, one in lying down and another in erect posture. Normally in erect posture, downward displacement of the kidney is seen. Downward displacement is not seen in case of perinephric abscess—*Mathe's sign*.

Renal Tuberculosis

Commonly it is *secondary*. Primary may be in the lung.

Pathological Types

Tuberculous papillary ulcer; Cavernous form; Hydronephrosis; Pyonephrosis [due to (secondary) superadded infection by *E.coli*, *Klebsiella*]; Tuberculous perinephric abscess; Calcified tuberculous area



Fig. 23.19: Stricture ureter in IVU in tuberculosis.

(mimics calculi, hence called as *pseudocalculi*); Caseous kidney—often called as *putty kidney* or *cement kidney* (it undergoes *autonephrectomy*); Miliary tuberculosis. Tuberculous bacilluria occurs from an early stage of the disease which causes tuberculous ureteritis and stricture ureter (**Fig. 23.19**). Tuberculous cystitis eventually results in *golffhole ureter* and *thimble bladder* (cystoscopic findings). Tuberculous prostatitis, seminal vesiculitis (P/R-palpable seminal vesicle), tuberculous epididymitis and funiculitis are other associations.

Clinical Features

Common in males; Common on right side; *Frequency*; *Polyuria*; *Sterile pyuria*. Urine is pale and opalescent with presence of pus cells without organisms in acid urine. **Abacterial aciduria**. (Other causes: Interstitial cystitis, chlamydia). *Painful micturition with often terminal haematuria*; Renal pain and suprapubic pain. *Tuberculous kidney is rarely palpable* unless there is hydronephrosis or perinephric abscess. Enlarged prostate and seminal vesicle, thickened beaded vas,

thickened epididymis, impotence, infertility, fever and weight loss, often cough with expectoration and haemoptysis are other features. *Three consecutive early morning samples of urine (EMSU)* are collected and sent for microscopy, Ziehl-Neelsen staining, culture (L-J media) or guinea pig inoculation. Plain X-ray KUB—shows calcification. IVU—Hydrocalyx, narrowing of calyx, stricture ureter which are multiple with dilatations in between. Often RGP is very useful, as better definition of ureter, pelvis, calyces and selective sampling of urine are possible.

Cystoscopy reveals multiple tubercles, bladder spasm, oedema of ureteric orifice eventually forming '*Golffhole ureter*', scarring, ulceration, bleeding, stone formation.

Hydronephrosis (HN)

It is an aseptic dilatation of pelvicalyceal system due to *partial or intermittent obstruction* to the outflow of urine.

Aetiology

It can be unilateral or bilateral.

Congenital PUJ is the commonest cause of HN. Often it is bilateral and presentation on one side is earlier than the other side. **Aberrant renal artery or vein** in the lower pole of kidney can compress the PUJ causing HN. Renal angiogram confirms the diagnosis. In *pregnancy* dilatation of ureters and both pelves occur due to atony of ureteric musculature by progesterone. It starts as early as in the first few weeks of pregnancy and lasts until few weeks after delivery. Involution occurs 2-12 weeks after delivery.

Classification

Unilateral HN; Bilateral HN without renal failure; Bilateral HN with renal failure.

Intermittent HN: Obstruction occurs; swelling and pain appear in the loin. After sometime patient passes large amount of urine following which swelling and pain disappear—*Dietl's crisis*. Persistent HN: It is due to persistent partial obstruction.

1. HN only. 2. HN with hydroureter.

1. Extrarenal pelvic HN (80%). 2. Intrarenal pelvic HN (20%)—Destruction of kidney is earlier and severe

Unilateral	Bilateral
A. Extramural: 1. Aberrant renal vessels (vein or artery) 2. Compression by growth [ca cervix, ca rectum] 3. Retroperitoneal fibrosis 4. Retrocaval ureter	A. Congenital: 1. Congenital stricture of external urethral meatus. Pin-hole meatus. 2. Congenital posterior urethral valve.
A. Intramural: 1. Congenital PUJ obstruction—<i>commonest</i> 2. Ureterocele 3. Neoplasm of ureter 4. Narrow ureteric orifice. 5. Stricture ureter following removal of stone, pelvic surgeries or tuberculosis of ureter.	
C. Intraluminal: 1. Stone in the renal pelvis or ureter. 2. Sloughed papilla in papillary necrosis.	

in case of intrarenal pelvic HN as compared to extra-renal pelvic HN.

Clinical Features

In unilateral cases: Congenital PUJ obstruction and calculus are the most common causes. M: F: 2:1. Right side kidney is affected more commonly. Presents with dull aching loin pain with dragging sensation or heaviness. Presents with mass in the loin which is smooth, mobile, ballotable, moves with respiration, dullness in renal angle with a band of colonic resonance in front. Attacks of acute renal colic dysuria, haematuria, if infected fever and tenderness in renal angle, occasionally hypertension are other features.

In bilateral cases: Lower urinary tract obstruction presents with loin pain; features of bladder outlet obstruction—frequency, hesitancy, poor stream. Kidneys are often not palpable if renal failure develops early. Bilateral upper urinary tract obstruction presents with loin pain, mass in the loin, attacks of renal colic. In bilateral case, when severe, features of renal failure like oliguria, oedema, and hiccough may be present.

Complications

Pyonephrosis, perinephric abscess, renal failure in bilateral cases.

Investigations

Blood urea and serum creatinine. Urine for microscopy.

US abdomen: Type of pelvis, thickness of parenchyma, site of obstruction and cause of obstruction (stones) can be made out.

IVU: To find out the function of diseased as well as opposite kidney. Normal calyx is cup-shaped. It gets *flattened* and later *club-shaped* which eventually becomes *broadened* in hydronephrosis (**Figs 23.20A to C**).

Whitaker Test

A fine needle is passed into the renal pelvis through loin. Pelvis is perfused with saline at a rate of 10ml/minute. Normally, initially the pressure rises and later it will become constant. Persistent increase in pressure suggests HN.

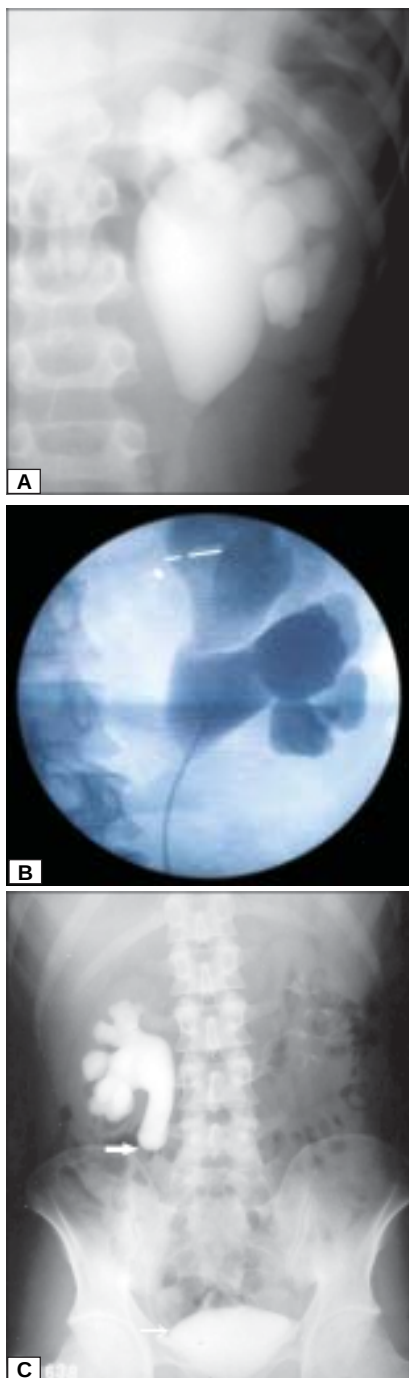
Isotope Renography

Isotope renography is also useful to study the function (DTPA) of the kidney before and after the surgical treatment and also to see the efficacy of surgery as far as function is considered (**Fig. 23.21**).

Pyonephrosis

It is collection of pus in pelvicalyceal system, which is converted into a multiloculated sac.

It occurs due to infection of preexisting hydronephrosis; following acute pyelonephritis or as a complication of renal calculus - either pelvic stone or staghorn calculus.



Figs 23.20A to C: IVU showing hydronephrosis in different patients. Note the dilated renal pelvis and delay in the excretion of the dye from the affected kidney. Second one is C arm photo.

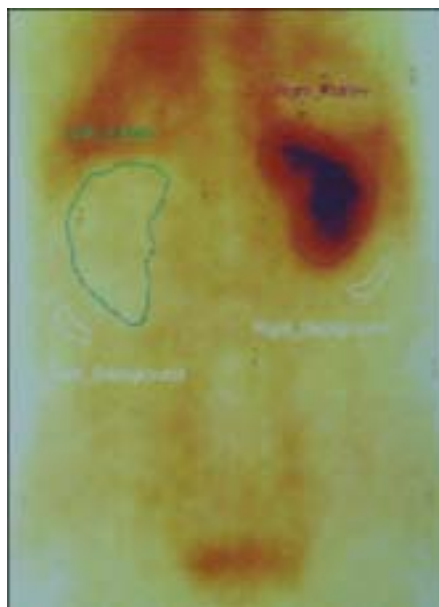


Fig. 23.21: Radioisotope scan (DTPA) of kidney showing hydronephrosis right sided.

Clinical Features

Triad—anaemia, loin swelling, pain. Present as tender mass in the loin which is smooth, soft, not mobile, not moving with respiration. Patient may also have cystitis, pyuria, burning micturition. Features of toxicity such as fever with chills and rigors may be seen.

Investigations

Plain X-ray KUB may show renal calculus. **IVU** shows HN. **Cystoscopy** reveals cystitis with *efflux of purulent pus* through the ureteric orifice. **US** shows dilatation.

Carbuncle of Kidney (Renal Carbuncle)

A localised inflammatory necrotic mass of tissue involving renal parenchyma, caused by *Staphylococcus aureus* and coliform organisms, source of which is cutaneous infections like boil and carbuncle. It presents as ill defined tender swelling in the loin, with pyrexia and leucocytosis. Staphylococci can be isolated from the urine. IVU shows obliteration of group of calyces, **mimicking renal cell carcinoma**.

Renal Calculus

It is more common in males; 90% are radio-opaque (Gallstones are more common in females; 90% are radio-luscent).

Aetiology

Diet: Vitamin A deficiency—It causes desquamation of epithelium which acts as a nidus for stone formation.

Climate: In hot climate urinary solutes will increase with decrease in colloids, which leads to chelation of solute with calcium forming a nidus for stone. **Citrate level** in urine (300-900 mg/24 hours) maintains the calcium phosphate and carbonate in soluble state and any decrease in citrate level in urine causes stone formation.

Infection in kidney: Urea splitting organisms commonly cause stone formation. *E. coli*, *Staphylococcus*, *Streptococcus*, *Proteus*. **Prolonged immobilisation** causes decalcification of bones and so hypercalciuria leading to stone formation. **Hyperparathyroidism** causes hypercalciuria causing multiple bilateral stones or often bilateral nephrocalcinosis. **Hyperoxaluria**, as a result of altered glycine metabolism. **Cystinuria** (Autosomal recessive). **Stasis** due to obstruction to urine flow.

Medullary sponge kidney: **Randall's plaque** theory is erosion and deposition of urinary salts as Randall's plaque at the apex of renal papillae. **Carr's postulates** states that minute concretions called as microliths normally develop in the subendothelial part of the tubule which will be carried away as particles by renal lymphatic network vessels. If these lymphatics are blocked microliths enlarge and act as nidus for stone formation.

Others: Sarcoidosis, myelomatosis, gout, idiopathic hypercalciuria, hypervitaminosis D, neoplasms on treatment, hypomagnesuria (Mg^{++} in urine acts as a complexing agent and prevents nucleation normally). **Renal tubular acidosis:** Commonly causes calcium phosphate stone. 10%.

Types

(1) **Oxalate stones** (75%): Also called as *mulberry stone* as it is brown in colour, with sharp projections. It is invariably calcium oxalate stone, shows *envelope*

shaped crystals in urine. (2) **Phosphate stones** (10-15%): It is either calcium phosphate or calcium, magnesium, ammonium phosphate stone usually occurring in an infected urine. It is smooth and dirty white in colour. In alkaline urine it enlarges rapidly, filling renal calyces taking their shape called as *staghorn calculus*. It is radio-opaque and attains a large size. (3) **Uric acid stones:** are smooth, hard, yellowish, multiple and radiolucent. They are seen in gout, hyperuricosuria, and altered purine metabolism. (4) **Urate stones.** (5) **Cystine stones** occur in *cystinuria* where there is defective absorption of cystine from the renal tubules (autosomal recessive condition). It is seen in young girls, occurs *only in acid urine*. It is multiple, soft, yellow in colour and the colour changes to *greenish hue* on exposure. It attains large size. It is radio-opaque because it contains sulphur. (6) **Xanthine stones** are very rare, smooth, brick red in colour, due to altered xanthine metabolism. Here there is deficiency in xanthine oxidase enzyme. (7) **Indigo stones:** Very rare. Blue in colour. (8) **Struvite stone:** It is compound of magnesium, ammonium, phosphate mixed with carbonate. It occurs in presence of ammonia and urea splitting organisms in urine.

Clinical Features

(1) **Pain:** Renal pain is located over renal angle, hypochondriac and lumbar region. Often it is severe, with vomiting due to pylorospasm. Often radiating to groin and testis. (2) **Haematuria** is common. (3) **Pyuria.** (4) **Fever.** (5) **Tenderness** in renal angle, with often palpable mass in the loin which is bimanually palpable, ballotable, smooth, soft, moves with respiration. (6) **As urinary tract infection.** (7) **Incidental finding.** (8) **Often hypertension.** Plain X-ray KUB shows radio-opaque stone; IVU is done to see the function; US is diagnostic; urine analysis and culture to identify the bacteria.

Ureteric Calculi

It is always of renal origin. Nature of stones is same as that of renal stones. They are commonly of elongated shape. They can get impacted at narrow sites at different levels—PUJ; where ureter crosses the iliac vessels; where ureter crosses vas deferens/broad ligament; where ureter penetrates outer layer of bladder muscle;



Fig. 23.22: Plain X-ray KUB showing stone in left ureter.



Fig. 23.23: Ureteric stone after extraction.

in the intramural portion of ureter near the ureteric orifice. Stones less than 5-8 mm size may pass spontaneously (**Figs 23.22 and 23.23**).

Problems with ureteric stones: Obstruction, hydronephrosis, infection, impaction, ureteral stricture.

Clinical Features

(1) *Pain*—colicky type, radiates from loin to groin often to the tip of the genitalia, testis. It is severe in intensity. It mimics *appendicitis, cholecystitis, ovarian or tubal pathology*. (2) Nausea, vomiting, sweating due to pain and reflex pylorospasm. (3) Haematuria,

dysuria, frequency, strangury. (4) Tenderness in iliac fossa and renal angle (no rebound tenderness).

Staghorn Calculus

It is the stone occupying the renal pelvis and calyces. It is usually phosphate or ammonium magnesium phosphate (*Triple phosphate*) stone. It is white in colour, soft, smooth, occurs in preexisting infection (commonly *E. coli*). It can be *unilateral or bilateral*. Patient with *bilateral* stones may go in for renal failure. Pain, haematuria, renal failure in bilateral cases, fever with chills and rigors are the presentations (**Fig. 23.24**).



Fig. 23.24: Staghorn calculi both sides in a plain X-ray.

Wilms' Tumour (Nephroblastoma)

It arises from embryonic connective tissue containing epithelial and connective tissue elements. It is located in one of the poles of the kidney. It is bilateral in 5% cases. It is common in first 4 years of life.

Gross: It is smooth, soft, fleshy, pinkish white in colour, often with haemorrhagic areas.

Microscopically: Malignant primitive glomeruli and primitive tubules, with epithelial and connective tissue cells exist side by side, one type is usually predominant.

Spread: Mainly through blood into the lungs, liver and rarely to bones.

Clinical Features

Triad: Mass, fever, haematuria. (1) *Mass abdomen* is commonest presentation. Mass is smooth, mobile,

firm or hard, lobular, located in the loin, moves with respiration, bimanually palpable, ballotable, with dullness in renal angle and with resonant band in front. It does not cross the midline. *Differential diagnosis* is adrenal neuroblastoma which is knobby and nodular, does not move with respiration and crosses the midline. (2) *Fever*: May be due to tumour necrosis. (3) *Haematuria* is a grave sign as it signifies rupture of tumour into the renal pelvis. (4) *Hypertension* (25%). (5) 12% of cases are associated with congenital anomalies and syndromes.

Investigations

US abdomen, IVU, renal angiography, X-ray abdomen—*egg shell* peripheral calcification is diagnostic, CT abdomen.

Differential diagnosis: *Adrenal tumour*, retroperitoneal tumour, renal cyst, polycystic kidney disease.

Staging of Wilms' tumour:

STAGE

- I. Disease confined to kidney and completely resectable.
- II. Tumour extends beyond kidney, but can be excised completely.
- III. Residual disease after resection with +ve lymph nodes and massive spillage.
- IV. Blood born metastasis.
- V. Bilateral disease.

Renal Cell Carcinoma (RCC)

Also known as *Hypernephroma*—(it is a misnomer), Grawitz tumour, clear cell carcinoma, Internist tumour. It is an adenocarcinoma arising from renal tubular cells—most common site is proximal renal tubular cell. More common in males; more common in 5th–6th decade of life.

Aetiology

It is associated with von-Hippel-Lindae disease (Cerebellar haemangioblastoma, retinal angiomas, tumour or cysts of pancreas). RCC here is commonly bilateral. High animal fat diet; environmental factors like asbestos, lead, cadmium and tobacco; cigarette smoking; chromosomal aberration; acquired cystic kidney disease after long term dialysis are some of the risk factors.

Pathology

Gross: It attains a large size. Commonly located in *upper pole*, sometimes in lower pole but rare in the middle. Cut section is *yellowish* due to lipoid content with areas of haemorrhage and necrosis. This noncapsulated tumour is very vascular.

Microscopy: Malignant cells which are cubical or polyhedral contain lipid, cholesterol and glycogen.

Spread

1. *Local*: Into the perinephric pad of fat, calyces and renal pelvis.
2. *Blood spread*: RCC enters the renal vein as *proliferating tumour thrombus* which extends into the IVC and later gets detached causing *cannon ball secondaries* in the lung which are often calcified. Once primary tumour is removed, secondaries may regress due to tumour immunity. Occasionally secondaries occur in bone, liver and brain. Left testicular vein which drains into left renal vein may get blocked by proliferating tumour thrombus resulting in *irreducible left sided varicocele*.
3. *Lymphatic spread*: To hilar lymph nodes, para aortic lymph nodes.

AJCC (American Joint Committee on Cancer) Staging: TNM staging.

- | | |
|-----|---|
| Tx | Primary tumour cannot be assessed. |
| T0 | No primary tumour. |
| T1 | Tumour less than 7.0 cm size, limited to kidney. |
| T1a | < 4.0 cm |
| T1b | 4–7 cm |
| T2 | Tumour more than 7.0 cm size, limited to kidney. |
| T3 | Tumour extends into major veins, adrenals, perinephric fat but not into the Gerota's fascia. |
| T3a | Into adrenal or perinephric tissue. |
| T3b | Into renal vein or IVC below diaphragm. |
| T3c | Tumour extends into IVC above the diaphragm. |
| T4 | Tumour invades Gerota's fascia and extends beyond. |
| N0 | No lymph nodes. |
| N1 | Spread to single regional lymph nodes. |
| N2 | Spread to more than one regional lymph nodes. |
| M0 | No blood spread. |
| M1 | Distant spread present – to lungs (75%); soft tissues (25%); bones (20%); liver (15%); brain; skin. |

Robson-Flocks Kadesky staging:

Stage 1	Tumour confined to renal parenchyma.
Stage 2	Tumour invasion to perinephric fat but confined with in Gerota's fascia
Stage 3	a. Tumour invasion to renal vein or IVC b. Invasion to regional lymph nodes c. Both a+b
Stage 4	Invasion to adjacent organs other than adrenal Distant metastasis

Clinical Features

M:F::2:1. *Triad*—Haematuria; pain; palpable mass. Others are clot colic; dragging discomfort in the loin; mass in the loin which moves with respiration, mobile, nodular, hard, with dull renal angle and resonant band in front.; left sided *varicocele* which is *irreducible*.

Atypical presentations: (A) Due to secondaries: pathological fractures, persistent cough and haemoptysis. (B) *Persistent pyrexia* with no evidence of infection (**Pyrexia of Unknown Origin**). (C) Constitutional symptoms: Malaise, lethargy and severe anaemia. (D) Polycythemia: 4%. (E) Hypercalcaemia, hypertension (Surgical renal conditions associated with hypertension-PCKD, Renal cell carcinoma, renal artery stenosis, chronic glomerulonephritis). (F) Nephrotic syndrome (very rare). (G) Stauffer's syndrome: Non-metastatic liver dysfunction which gets corrected after nephrectomy.

Investigation

Urine microscopy for RBCs. IVU—shows mass lesion and irregular filling defect. US abdomen—To know the size, extension, lymph node involvement, spread to the liver, status of renal vein and IVC. CT scan: It is confirmatory and also helps to know the status of renal vein and IVC. Renal angiogram is done through Seldinger technique via transfemoral route, to see the vascularity. *Pharmacangiogram* (Inject noradrenaline along with dye while doing angiogram) - As tumour vessels are autonomous they will not constrict whereas adjacent normal vessels will constrict, so *tumour blush* is visualised. Through angiogram therapeutic embolisation of tumour can be done to reduce the vascularity of tumour. Chest X-ray shows *cannon ball* secondaries. Often it is *calcified*. CT chest and bone scan are done to look

for bone secondaries. Peripheral smear, serum calcium, haematocrit and ESR.

Differential Diagnosis

Polycystic kidney disease; solitary cyst of kidney; adrenal tumour; retroperitoneal tumour; carcinoma colon.

Ectopia Vesicae (Exstrophy of the Bladder)

It is incomplete development of the infraumbilical part of the anterior abdominal wall and anterior wall of the bladder. It is of embryological origin. It is often associated with the spina bifida and other congenital anomalies. It is more common in males (4:1). Red mucus membrane of posterior bladder wall protrudes out with visible urine efflux from ureteric orifice. Umbilicus is absent. There is separation of pubic bones. In males, *epispadias* is commonly present with rudimentary prostate and seminal vesicle. Testis is normal. Bilateral inguinal hernias are common. In females defective external genitalia is common.

Problems: Repeated soakage; ulceration; pain; recurrent pyelonephritis; renal failure; metaplastic changes in mucosa can lead into *adenocarcinoma*. 50% of patients die of renal failure.

Vesical Calculus**Types**

Primary vesical calculus: It occurs in sterile urine. It usually comes down from kidney through ureter into the bladder and there it gets enlarged. It is usually *oxalate* stone. Oxalate stone is usually single, primary stone, brownish black in colour (due to deposited blood pigment over the surface), hard and with spikes over the surface which irritates bladder mucosa causing haematuria (*mullberry stone*).

Secondary vesical calculus: It occurs in the presence of infection. It is usually phosphate *stone*, occurs in bladder only. Phosphate stone is smooth, soft, ivory white in colour. It is either calcium phosphate or ammonium, calcium and magnesium phosphate (*Triple phosphate stone*).

Uric acid and urate stones are single or multiple, primary, nonradio-opaque, smooth, pale yellow in colour.

Cystine calculus: Occurs in cystinuria and is radio-opaque due to high sulphur content.

Aetiology

1. Infection
2. Hypercalciuria of any cause.
3. Hyperoxaluria.
4. Cystinuria.
5. Bed ridden and paraplegic patients.
6. Gout and other hyperuricaemic patients.
7. Diverticula bladder.
8. Obstruction to urine flow by BPH, urethral stricture, bladder neck obstruction.
9. Neurogenic bladder.
10. Schistosomiasis.
11. Foreign body in bladder.

Clinical Features

Common in males. Often occurs in children. *Frequency* is more during day time than during night, because during the day, due to ambulation stone comes in contact with the trigone of the bladder and irritates, whereas during night, stone slips towards the fundus, away from the trigone and so less frequency and pain.

Pain: More during day which is referred to the tip of penis or labia. Also increases during jolting and movements. Suprapubic pain and tenderness may be present.

Haematuria: Often terminal. Interruption of urinary stream and often acute urinary retention. Features of cystitis: Burning micturition, fever, pain. P/R or P/V: Large stone may be palpable. Stone may be identified incidentally in plain X-ray KUB or US abdomen.

Investigations

Urine Microscopy

Envelope crystals in oxalate stone, hexagonal type in cystine stone. Other investigations are urine culture, blood urea, serum creatinine, serum calcium, inorganic phosphate, uric acid. Plain X-ray KUB shows radio-opaque stones—90%. IVU to see function of the kidney. US abdomen is diagnostic. Cystoscopy to see radiolucent stone (**Figs 23.25 to 23.27**).

Cystitis

It is inflammation of the bladder mucosa due to different causes.



Fig. 23.25: Typical oxalate stone—brown coloured with spikes.



Fig. 23.26: Oxalate and phosphate stones in two different patients.



Fig. 23.27: Plain X-ray KUB showing radio-opaque bladder stone – phosphate stone.

Causes

Acute bacterial cystitis; Chronic cystitis due to tuberculosis, syphilis; interstitial cystitis; radiation cystitis; cystitis due to schistosomiasis; post menopausal atrophic cystitis.

Predisposing Factors

Congenital urinary anomalies; Short urethra in females may cause ascending infection and cystitis; initial period of sexual contact in females can cause cystitis—*Honeymoon cystitis*; Catheters, instrumentation; bladder stone; BPH, carcinoma prostate; cystocele, bladder diverticulum; stricture urethra, bladder neck obstruction; bladder tumours; pregnancy; CNS diseases, spinal injury. *Organisms: E.coli, Klebsiella, Pseudomonas, Staph aureus, Staph albus, Proteus; Candida albicans*, fungal infection.

Features

Often painful urination, frequency, strangury, incomplete emptying, retention. Occasionally haematuria can occur. Burning urine, discoloured foul smelling urine, fever, chills, rigors, suprapubic pain and tenderness and often loin pain are other features. Septicaemia can develop in severe cystitis.

Interstitial Cystitis (Hunner's Ulcer, Elusive Ulcer)

It is common in western psychic females.

Pathology: There is pancystitis with fibrosis of the vesical musculature, with linear ulcers in the bladder mucosa. Microscopically, severe inflammation of all layers of bladder with fibrosis is observed. Bladder eventually becomes **thimble (systolic) bladder** with decreased bladder capacity up to 30-60 ml (less than 100 ml).

Clinical Features

Pain, decreased bladder capacity, pain increases with bladder distension, frequency and often haematuria.

Schistosoma Haematobium (Endemic Haematuria, Urinary Bilharziasis)

Life Cycle

Fresh water snail – (Intermediate host). Bifid tailed embryos (cercariae) in infected water → Penetrate the skin → through circulation enters the liver → Male and female worms form → Sexual fusion and maturity occurs → Flows through the portal vein in retrograde direction along the inferior mesenteric vein → Vesical venous plexus → reach bladder wall and submucosa → Ova are released → Enters the bladder through the mucosa → Ova are released into the urine and to water → Ciliated miracidium → Fresh water snail → Death of snail releases thousands of cercariae.

Pathology in the Bladder

Bilharzial pseudotubercles –earliest sign;. nodules; sandy patches – calcified dead ova with degeneration of overlying epithelium; granulomas; ulceration and papilloma; fibrosis and thimble bladder formation. In due course of time, *squamous cell carcinoma* can develop. Other pathologies: Ureteral and urethral stricture, recurrent UTI, bladder calculi, urinary fistula, etc.

Clinical Features

Initially cutaneous lesions like urticaria develop which lasts for few days. Then after a period of 4-8 weeks, fever, along with features of eosinophilia occurs. Eventually after many months it causes intermittent, painless, *terminal haematuria*.

Differential Diagnosis

Tuberculosis, recurrent cystitis, malignancy.

Thimble or Systolic Bladder

It is inability of the bladder to relax and distend and so unable to retain the urine as required.

Causes

Tuberculous cystitis; *Schistosoma haematobium*; Interstitial cystitis; Radiotherapy; Malignancy; Previous bladder surgery. Bladder is fibrotic and

contracted with difficulty in dilating and accommodating urine as needed. Bladder capacity will be less than 100 ml (60 ml).

Clinical Features

Decreased bladder capacity; Frequency; Features of recurrent cystitis.

Investigations

Cystoscopy, Cystography, IVU, Urine C/S, Specific diagnostic tests.

Bladder Tumours

(1) Primary: (a) Epithelial: *Transitional cell carcinoma* (90%); *Adenocarcinoma*, arising from urachal remnant or in exstrophy bladder or from glandular metaplasia (2%); *Squamous cell carcinoma* originates from bilharzial infection (5%) or calculus.

(b) Connective tissue tumour: Myoma, angioma, fibromas, sarcomas, extra adrenal pheochromocytoma. **(2) Secondary:** From adjacent organs like sigmoid colon, rectum, uterus, ovary, prostate.

Transitional Cell Carcinoma (TCC)

It is the commonest type of bladder tumour.

Aetiology: 3C's—chemical carcinogens; cigarette smoking; cyclophosphamide. Chemical carcinogens are the main factor—2-Naphthylamine, aminobiphenyl, benzidine, chloro-O-toluidine, chloroaniline, and other dyes. Occupation wise it is common in textile, dye, cable, tyre, petrol, leather workers, painters, chemical workers, sewage workers.

Tumour groups: (a) *Non muscle invasive tumour without involving lamina propria*: It has got excellent prognosis (70%). (b) *Non muscle invasive tumour with involvement of lamina propria*. (c) *Muscle invasive type* (125%)—Carries poor prognosis. (d) *Carcinoma in situ* (flat noninvasive)—Contains irregularly arranged cells with large nuclei, with high mitotic index, replacing normal urothelium. This may occur alone—*Primary carcinoma in situ*. It may occur in association with a new tumour—*Concomitant carcinoma in situ*. It can occur in a patient who had a previous tumour

—*Secondary carcinoma in situ*. It has got high malignant potential with 50% mortality rate. It was earlier called as *malignant cystitis* as it causes severe dysuria, suprapubic pain and frequency.

Types of bladder tumours: (a) **Superficial bladder tumour:** It may be *papillary*, pedunculated with narrow stalk, which is often multiple. It may be *sessile* with a wide base, which can be single or multiple, and has got tendency to invade the muscle earlier. Mucosa in and around the tumour is oedematous, red, with dilated vessels, often with encrustations. (b) **Muscle invasive TCC:** Almost always they are solid, sessile, with a broad base and with irregular ulcerated surface. It may spread through lymphatics to pelvic lymph nodes or through 'blood to the lung, liver and bones'. It has got poor prognosis. (c) **Carcinoma in situ.**

Sites: **Lateral wall**—Commonest (35%). Trigone—next common (32%).

Jewett-Strong-Marshall staging

- I. Subepithelial connective tissue.
- II. Muscle infiltration superficially.
- III. Full thickness muscle and perivesical tissue infiltrated, but mobile.
- IV. Fixed to adjacent organs
 - IVa. prostate
 - IVb. pelvic wall

Staging is done by bimanual palpation under G/A.

Clinical Features

(1) Painless haematuria. (2) Features of cystitis, with suprapubic pain, frequency, dysuria. (3) Hydronephrosis can occur when tumour obstructs the ureteric orifice. (4) Pain in groin, back, perineum, when tumour invades the pelvic wall.

Investigations

Urine microscopy: for RBC's and malignant cells. Blood: Hb%, blood urea, serum creatinine. IVU: shows filling defect with distortion and often hydronephrosis. Cystoscopy is diagnostic. *Bimanual examination under G/A*—to stage the tumour. US abdomen is done to see extension into bladder wall, pelvis, liver, lymph nodes. CT scan is done to evaluate the level of extension.

Residual Urine

It is the amount of urine retained in the bladder after voiding (after completion of the act of micturition). Normal value is 30 ml. Amount more than 50 ml is significant. It signifies obstruction in the urethra like BPH. Residual urine more than 200 ml in BPH indicates the need for surgical intervention. High residual urine is also seen in different types of neurogenic bladder. Residual urine precipitates infection because of stasis.

Residual urine is assessed by following methods:

The patient is asked to pass urine and then a red rubber catheter is passed to empty the retained urine which will be measured to quantify. It can also be evaluated by ultrasound or intravenous urogram - post micturition film (**Fig. 23.28**).

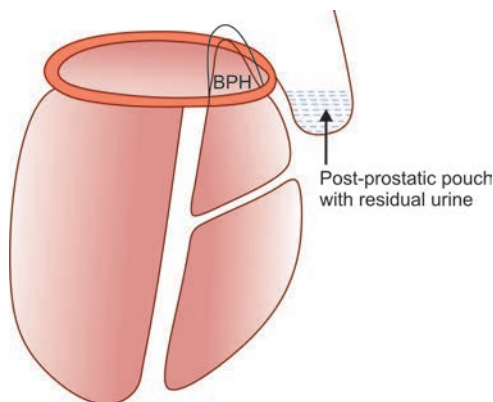


Fig. 23.28: Residual urine in post-prostatic pouch.

Malakoplakia

It is usually associated with chronic cystitis of unknown aetiology causing raised grayish patches in the bladder mucosa. Microscopically it shows infiltration of submucosa with lymphocytes, plasma cells and large multinucleated malakoplakia giant cells with concretions called as **Michaelis Gutmann bodies** both inside the giant cell as well as outside. It does not turn into malignancy.

Acid Phosphatase

It is the enzyme that splits organic phosphates. It is found in many human tissues, but more concentrated in prostate. It is active at pH 5. Acid phosphatase

secreted by prostate drains into the urethra through prostatic ducts and so blood levels of this enzyme remains low. Serum acid phosphatase estimation should be done **in empty stomach** because heavy meals can alter the level of the acid phosphatase. Normal value is *0-5 King Armstrong units per 100 ml of serum*. It is raised significantly in carcinoma prostate with metastases. *It does not increase in BPH*. Slight increase in acid phosphatase level occurs in acute prostatitis, Paget's disease of bone and hepatic cirrhosis. Prostatic fraction of acid phosphatase is more relevant in carcinoma prostate. Osteosclerotic osseous metastases in carcinoma of prostate are due to alkaline phosphatase.

Prostate Specific Antigen (PSA)

It is a protease produced from the prostatic epithelium secreted in the semen to cleave and liquefy the seminal coagulum formed after ejaculation. PSA is organ specific. *Normal value is 4 ng/ml of plasma. More than 10 ng/ml is significant*. PSA elevation occurs not only in carcinoma but also in prostatic hyperplasia and prostatitis. But level of increase is much more in carcinoma than in benign conditions. PSA occurs in two forms: *Major bound form and minor free form*. Major bound form increases in carcinoma. Minor free form is increased in benign conditions. PSA density, i.e. PSA level per gram of prostate tissue is more relevant. Serial estimation of PSA is very useful to suspect spread and recurrence after treatment. 25% of men with PSA 4-10 mg% show prostate carcinoma. 20% of men with normal PSA (1-4 mg %) will show prostate carcinoma. PSA more than 10 mg% is suggestive of carcinoma prostate. PSA more than 35 mg% is almost diagnostic of advanced carcinoma of prostate. Decrease in PSA after therapy suggests adequate ablation.

Benign Prostatic Hyperplasia (BPH)

It is benign enlargement of prostate which occurs after 50 years, usually between 60-70 years.

Theories

(1) It is involuntary hyperplasia due to a disturbance of the ratio and quantity of circulating androgens and estrogens. (2) BPH is a benign neoplasm called as **fibro-myo-adenoma**.

Pathology

BPH usually involves median and lateral lobes or one of them. It involves adenomatous zone of prostate, i.e. submucosal glands. Median lobe enlarges into the bladder. Lateral lobes narrow the urethra causing obstruction. *Urethra above the verumontanum* gets elongated and narrowed. *Bladder* initially takes the pressure burden causing *trabeculations, sacculations* and later *diverticula* formation. Enlarged prostate compresses the prostatic venous plexus causing congestion termed as *vesical piles leading to haematuria*. Incrimination of BPH as the source of haematuria before excluding other causes is termed as **Decoy prostate**. *Kidney and ureter*: Backpressure causes *hydroureter and hydronephrosis*. Secondary ascending infection can cause *acute or chronic pyelonephritis*. Often severe obstruction can lead onto obstructive uropathy with *renal failure*. BPH causes *impotence*.

Clinical Features

Frequency occurs due to introversion of sensitive urethral mucosa into the bladder or due to cystitis and urethritis. Other features are urgency; overflow and terminal dribbling; difficulty in micturition with weak stream and dribble; pain in suprapubic region and in loin due to cystitis and hydronephrosis respectively; acute retention of urine; retention with overflow; haematuria; renal failure. *Prostatism* is a combination of symptoms like frequency both at day and night, poor stream, delay in starting and difficulty in micturition. Tender area in suprapubic region with palpable enlarged bladder due to chronic retention is also seen. Hydronephrotic kidney may be palpable. Per rectal examination shows enlarged prostate. It should be done when bladder is empty. Features of urinary infection like fever, chills, burning micturition, etc. are also seen.

Differential Diagnosis

Stricture urethra; bladder tumour, carcinoma prostate; neurological causes of retention of urine like diabetes, tabes, disseminated sclerosis, Parkinson's disease.

Investigations

Urine for microscopy and C/S; Blood urea and serum creatinine; US abdomen; residual urine assessment;

urodynamics; cystoscopy; acid phosphatase; prostate specific antigen (PSA); IVU; Serum electrolytes.

Carcinoma Prostate

It is the *commonest malignant tumour* in men after 65 years. Carcinoma prostate occurs in peripheral zone in prostatic gland proper, i.e. *commonly in posterior lobe*. So prostatectomy for BPH does not confer protection against development of carcinoma prostate.

Types of Carcinoma Prostate

(a) *Microscopically latent*—Tumours incidentally found either by TURP or by PSA estimation. (b) *Early localized carcinoma*. (c) *Advanced local prostatic carcinoma*. (d) *Metastatic carcinoma* either into the bone commonly or other organs.

Histology

It is an *adenocarcinoma*, where there is loss of the myoepithelial cell layer which normally surrounds the prostatic glands (Gleeson). Glands here appear in confluence. Grading of carcinoma is based on dedifferentiation as proposed by Gleeson.

Staging of Carcinoma Prostate

Occult—Diagnosed after investigation due to suspicion.
Stage I – Tumour confined to prostate/local nodule.
Stage II – Tumour involving capsule or diffuse type.
Stage III – Tumour involving seminal vesicle.
Stage IV – Extension into adjacent tissues.

Local spread: Upward into seminal vesicles, bladder neck, trigone, later into both ureters causing anuria, etc. Downward extension into distal sphincter.

Blood spread: Into the bones commonly - pelvic bones, lumbar vertebrae, femoral head, ribs, skull in that order. Secondaries in bone are *osteoblastic* due to serum alkaline phosphatase. Pathological fractures can occur in long bones and vertebrae. Paraplegia may occur if spine is involved. Rarely spread to liver and lung can occur.

Lymphatic spread: Into the obturator lymph nodes, then to internal iliac lymph nodes. Through seminal vesicles, spreads into external iliac and retroperitoneal

lymph nodes. Eventually mediastinal, and left supraclavicular lymph nodes get involved.

Clinical Features

Commonly asymptomatic. May present with bladder outlet obstruction and so retention of urine. Other features are haematuria, frequency, pelvic pain, back pain, arthritic pain in sacroiliac joint. On per rectal examination, prostate feels hard, nodular, and irregular often with loss of median groove. It may be incidental carcinoma after TURP or after PSA analysis. It may present with features of renal failure, anaemia which may be secondary to extensive bone marrow invasion or due to renal failure.

Differential Diagnoses

Differential diagnoses are other causes of retention of urine and other causes of back pain.

Investigations

Hb%, Peripheral smear. Prostatic specific antigen (PSA): More than 10 nmol/ml is diagnostic. Prostatic fraction of acid phosphatase is increased. Blood urea, serum creatinine, liver function tests. Trans Rectal Ultra Sound (TRUS) is very useful. Transrectal prostatic biopsy. Plain X-ray KUB may show **dense coarse sclerotic** secondaries. Osteolytic or combination of lytic and sclerotic lesions are also often seen (**Fig. 23.29**). Technetium radioisotope bone scan to see

secondaries. US abdomen to see the extension into the bladder and to look for hydronephrosis in kidneys.

Note: Osteoblastic secondaries are also occasionally seen in carcinoma breast.

Prostatitis

It can be acute or chronic. It is often very distressing condition and also often difficult to diagnose. **Acute** may be due to instrumentation, ascending/descending infection. *E. coli*, streptococci, gonococci are common bacteria involved. Pain, frequency, fever, retention of urine, perineal heaviness, pain in defaecation are the features. Rectal examination feels tender firm prostate. Initial part of urine is turbid. It may lead into prostatic abscess (single/multiple), chronic prostatitis, retention of urine. **Chronic prostatitis** is associated with posterior urethritis, epididymitis. Back pain, perineal pain, leg pain, fever, sexual dysfunction are common. Prostatic massage extracts prostatic gleet (early morning massage fluid) for cytological analysis. In three glass urine test, first glass shows *prostatic threads*.

Prostatic abscess: It is infection, suppuration and pus formation in the prostate gland. Present with fever, rigors, perineal pain, urinary disturbances, and tender soft fluctuant swelling in the prostate on rectal examination. Often presents with retention of urine. Total count will be increased. Urine will show pus cells. US is diagnostic and is often done over perineum also.

Stricture Urethra

Classification I: Etiologically: (1) Congenital. (2) Inflammatory: (a) Post-gonococcal is commonest (70%). Common in the bulb of urethra especially in the roof. Here multiple strictures are common. Proximal stricture is the narrowest. (b) Tuberculous. (c) Other infection (Urethritis). (3) Traumatic—common, may be bulbous/membranous. (4) Post-instrumentation: Catheter, dilator, cystoscope. (5) Post-operative: Prostate surgery, urethrostomy.

Classification II: (1) Proximal: Common in *bulbous urethra* (70%). (2) Distal: Congenital (in the external meatus). Often traumatic in children.

Classification III: (1) Permeable—permits urine to pass. (2) Impermeable.

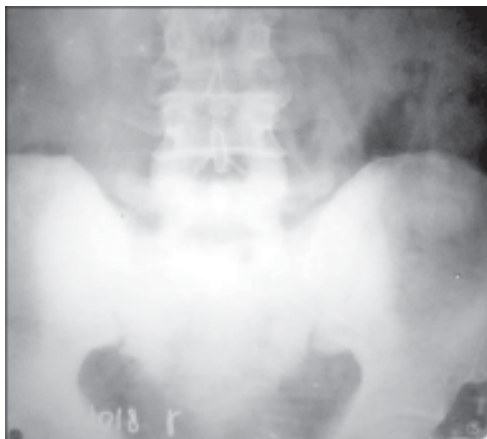


Fig. 23.29: A plain X-ray AP view of lumbar spine and pelvis in a patient with carcinoma of prostate. Note the typical osteosclerotic secondaries.

Classification IV: (1) Passable: Allows catheter to pass. (2) Impassable.

Classification V: It can be single or multiple.

Classification VI: According to parts involved. In the roof (commonest) or in the floor.

Clinical Features

(1) Poor urinary stream. (2) Forking and spraying of the stream. (3) Incomplete emptying. (4) Frequency, dysuria. (5) Retention and often with overflow. (6) Pain, burning micturition, suprapubic tenderness. (7) Thickening and button-like/crescentic feeling in bulbar urethra (Bulbous urethra is felt clinically by lifting the scrotum in midline in the perineum).

Investigations

US abdomen, X-ray of pelvis to see old fracture with history of trauma. *Ascending Urethrogram* is an essential investigation—to see the site, type, extent and false passage. The dye is injected through suprapubic needle puncture into the bladder and visualisation is done using C-ARM image intensifier (**Fig. 23.30**). Urodynamic studies and Urethroscopy are other investigations done.



Fig. 23.30: Ascending urethrogram showing failure of dye to pass due to stricture urethra.

Complications of Stricture Urethra

Retention of urine; urethral fistula; Infection—urethritis, cystitis, pyelonephritis; urethral diverticula; peri-

urethral abscess; bilateral hydronephrosis; stone formation; renal failure; Due to straining—hernia, haemorrhoids, rectal prolapse.

Posterior Urethral Valve

They are congenital symmetrical valves in the posterior urethra just below the verumontanum. It allows the passage of catheter without obstructing its ingress. But it obstructs the outflow of urine. Proximal urethra is enormously dilated with obstructive pathology in the bladder (sacculations and diverticula formation). Bladder wall is thickened and hypertrophied so much so that it is palpable in suprapubic region as firm swelling—*Cricket-ball bladder*. There is poor urinary stream, with hydronephrosis, often with infection. Child finds difficult to pass urine. Often it is associated with vesicoureteric reflux. *Micturating cystourethrography (MCU)* is diagnostic. It shows dilated proximal urethra.

Differential Diagnosis

(1) *Marion's disease*: Bladder neck obstruction due to hypertrophied inter-ureteric bar. MCU differentiates it from posterior urethral valve. (2) Neurogenic bladder.

Urethral Calculi

Stone from the bladder is commonly passed out through urethra if it is small, but the stone can get impacted due to a *stricture or urethral diverticulum*.

Sites of Impaction

Prostatic urethra, bulbous urethra, fossa navicularis, external meatus.

Clinical Features

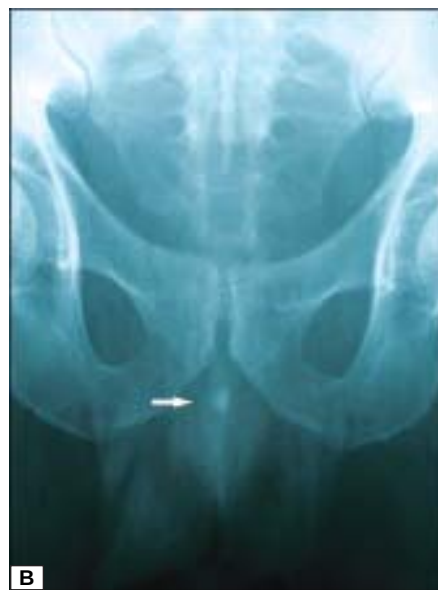
Painful urination with thin stream and forking of urine. Other features are retention of urine, lower urinary tract infection, haematuria, pain in the penis and perineum. Stone may be palpable when it is in the bulbar or penile urethra (**Figs 23.31A and B**).

Complications

Bleeding, stricture urethra and infection.



A



B

Figs 23.31A and B: Plain X-ray showing urethral stone.

Urethritis

Causes

Gonococcal urethritis/Non-gonococcal urethritis: Trauma—catheters, cystoscopes, stones. Trichomonal urethritis. Lymphogranuloma venereum (LGV). Chemical urethritis, Mycoplasma, Reiter's disease—Arthritis, conjunctivitis, urethritis, allergy.

Clinical Features

Urethral discharge, dysuria, burning micturition, haematuria and increased frequency are the presenting features. Others are perineal pain, tenderness over the site, suprapubic pain and tenderness.

Urinary Fistulas

It is the leak of urine at abnormal sites.

Causes

Congenital—Patent urachus.; Ectopiae vesicae.; In association with anorectal malformation—rectovesical fistula.

Acquired—Trauma to perineum., pelvic surgery. *Vesicovaginal fistula*—due to obstructed labour; during hysterectomy bladder may get injured while mobilising it down; anterior colporrhaphy in elderly after vaginal hysterectomy; radiation induced; infiltrating carcinoma cervix. Usually it takes one week to present following necrosis and fistula formation. Features are – passage of urine per vagina, dribbling of urine in vagina on per speculum examination, swab test will be positive (methylene blue is injected into urethra after placing a swab in the vagina. Swab turns blue if there is fistula). Specific causes like tuberculosis, staghorn calculi can cause fistula after nephrostomy. Fistula can develop after surgery of renal pelvis, ureter, and bladder. If there is distal obstruction like stricture urethra, it will persist otherwise it subsides spontaneously. Renal pelvis is involved by Crohn's disease.

Examination of Acute Abdomen

It is sudden severe attack of abdominal pain to such an extent that patient is in severe agony and often in shock. Many of the conditions causing acute abdomen may be life threatening like perforation of intestine. Proper clinical examination is the essential step in these patients to conclude the diagnosis and to plan the therapy.

CAUSES OF ACUTE ABDOMEN

INTRA-ABDOMINAL CAUSES

Inflammation—Acute appendicitis, acute cholecystitis, acute salpingitis, acute diverticulitis, acute Crohn's, acute mesenteric adenitis, primary acute peritonitis.

Perforation of bowel

Acute intestinal obstruction—In the lumen (roundworm, gallstones), in the wall (stricture, intussusception, tumour), outside wall (hernia, bands, adhesions, volvulus).

Mesenteric vessel occlusion by thrombosis or embolism.

Haemorrhage—Ruptured ectopic gestation, ruptured tropical spleen like malarial, ruptured aortic aneurysm.

Torsions—Twisted ovarian cyst, twisted splenic pedicle.

Colicky causes—Ureteric, biliary, intestinal, appendicular.

EXTRA-ABDOMINAL CAUSES

In the abdominal wall—Abdominal wall abscess, Meleney's spreading gangrene, rupture of abdominal wall muscles, inferior epigastric artery tear and haematoma formation. **In thorax**—Lobar pneumonia, diaphragmatic pleurisy, pericarditis, angina pectoris, coronary disease.

Retroperitoneal causes—Acute pyelonephritis, retroperitoneal lymphadenitis and lymphangitis, ruptured aortic aneurysm.

Diseases of spine, spinal cord and intercostal nerves—Pott's tuberculous spine, gastric crisis of Tabes dorsalis, herpes zoster of intercostal nerves with neuralgia.

Other causes—Malaria, typhoid, porphyria, diabetic crisis, sickle cell disease, purpura, haemophilia, etc.

History

Age

Sigmoid volvulus, carcinoma colon causing obstruction, diverticulitis are common in old people. Acute pancreatitis, acute cholecystitis, perforation are common in adults. Appendicitis is common in young adults. Roundworm obstruction is common in children. Midgut volvulus, intussusception is common in infants. Intestinal atresia, meconium ileus, anorectal malformation are common in newborn.

Sex

Ruptured ectopic gestation, twisted ovarian cyst are seen in females. Acute cholecystitis, primary peritonitis are common in females. Volvulus, intussusception, perforated peptic ulcer are common in males.

Residence

Perforation is more common in India where diet is rich with spicy foods. Acute cholecystitis is common in Bihar and north east India. Acute pancreatitis is common in Kerala and in Western countries.

Socioeconomic Group

Perforation, roundworm obstruction is more common in lower socioeconomic group; appendicitis is more common in higher socioeconomic group due to high protein low fibre diet intake.

History of Present Illness

Pain

Pain is *the most common* presentation of acute abdomen.



Fig. 24.1: Patient is asked to point out the site of pain at one point – pointing test.

Site of pain should be confirmed by asking the patient to point at one place using his finger – *pointing test* (**Fig. 24.1**). Pain is in right hypochondrium in acute cholecystitis; in right iliac fossa in acute appendicitis; in the loin in urinary stone; in epigastrium in acute pancreatitis.

Mode of onset of pain: It is sudden in onset in perforation, stones, torsion. Pain is initially less severe gradually progressive and becomes more in acute intestinal obstruction. Pain in acute appendicitis starts in early morning, initially boring and vague but later becomes severe and localized. Duodenal ulcer perforation follows afternoon food and becomes severe in the evening or night. Straining increases the pain in perforation; movements or jolting increases the colicky pain of ureteric stone.

Change in position of pain or spread of pain: In acute appendicitis pain initially begins at umbilicus but later shifts to right iliac fossa. Initial visceral pain occurs in and around umbilicus due to same segmental nerve supply (T_{10}); later pain in right iliac fossa is due to parietal peritonitis. In duodenal ulcer perforation pain initially begins in epigastrium but spread of peritonitis occurs and so also the pain when once gastric contents spills over into the right paracolic gutter. In acute pancreatitis, pain begins in epigastrium and radiates to back. Pain is said to be referred if it occurs at the same segmental cutaneous distribution of the nerve. Visceral part is sympathetic supply of the seg-

ment whereas the cutaneous part is somatic. Examples are—diaphragmatic irritation due to subphrenic abscess, inflammation, clot causes pain in skin over shoulder through C_3 , C_4 through phrenic nerve and supra-clavicular nerve. Head down position will aggravate the pain. Gastroduodenal and jejunal diseases have referred pain to epigastrium (T_{5-8}); ileum and appendix to umbilical region (T_9 , T_{10}); colon to hypogastrium ($T_{11,12}$, $L_{1,2}$); ureteric colic shows pain from loin to groin to scrotum and upper inner part of thigh through genitofemoral nerve ($L_{1,2}$). Gallstone colic refers from right hypochondrium to inferior angle of scapula (T_{7-9}). Pleuritis, haemothorax, pneumothorax causes referred pain to the abdominal wall mimicking acute abdomen.

Type of pain; aggravating and relieving factors:

Colicky pain is gripping in nature which appears occurs and disappears suddenly occurs due to spasm of hollow viscus like intestine (intestinal colic), ureter (ureteric colic), and common bile duct (biliary colic). Throbbing pain of cholecystitis, severe pain of acute pancreatitis, continuous burning constant pain of peritonitis are other types of pain. Pain may be initially colicky type but later becomes severe and continuous as where initial appendicular colic becoming persistent pain of obstructive appendicitis. Colicky pain is *relieved* by pressure; inflammatory pain gets *aggravated* by pressure. In cholecystitis, appendicitis, ureteric stone, pain aggravates by movements and jolting. Pain of diaphragmatic irritation aggravates on coughing, and deep breathing. Pain is relieved if patient avoids any movements like in acute peritonitis (silent/still abdomen); if patient vomits like in duodenal ulcer perforation, and colicky pain of ureteric stone; if sits and leans forward in acute pancreatitis (**Fig. 24.2**).

Vomiting

Character of vomiting whether projectile or regurgitant; *frequency* of vomiting (repetitive and profuse in acute pancreatitis and acute intestinal obstruction; periodical and infrequent in duodenal ulcer perforation); *quantity* and nature of the vomitus should be asked. *Vomitus* may be gastric, bilious, intestinal, faeculent or blood stained. History of haematemesis is also important. Vomiting is the *early* feature in proximal intestinal obstruction (jejunal); it is *late*



Fig. 24.2: Patient leans forward in acute pancreatitis to relieve pain.

feature in distal obstruction (terminal ileum or colon). Vomiting develops *after* feeling of pain in acute appendicitis, pancreatitis, colics. Pain and vomiting appears *simultaneously* in intestinal obstruction.

Pain first; vomiting next; fever last—feature of acute appendicitis—*Murphy's syndrome*.

Bowel Habits

Appendicitis can cause diarrhoea if pelvic appendix irritates the rectum. Appendicitis also may cause colonic spasm leading into constipation. Absolute constipation occurs in acute intestinal obstruction which means neither faeces nor flatus can be passed. Bloody putrid stool is seen in mesenteric ischaemia. Diarrhoea may be a feature of acute ulcerative colitis, enteritis. Passing blood and mucus with distension is a feature of acute intussusception.

Abdominal Distension

Patient often presents with fullness of abdomen which is gradually progressive and is associated with constipation and vomiting.

Urinary Symptoms

Burning urine, frequency, strangury can occur in retrocaecal/pelvic appendicitis, pelvic peritonitis due to irritation of ureter or bladder.

Other History

Fever, chills and rigors are important in acute abdomen. History of jaundice may be significant in acute cholecystitis, pancreatitis.

Past History

Past history of laparotomy is important in intestinal obstruction. Earlier history of renal stone disease, appendicitis treated with drugs, biliary colic, jaundice, pancreatitis, periodicity of pain in duodenal/gastric ulcer, haematemesis or melaena in the past should be asked for. Past history suggestive of acute abdomen and hospitalization is important.

Personal History

Missed menstrual cycles may suggest ectopic pregnancy in females. Urine pregnancy test and US abdomen may confirm the diagnosis. Smoking, alcohol intake, diet history are important.

General Examination

General look: Typical *facies hippocratica* of terminal stage of peritonitis is explained as anxious look, sunken bright eyes, pinched face, and cold sweat. Signs of *dehydration* like sunken eyes, dry tongue, and drawn cheeks are observed. Severe sudden *pallor* with shock in a female may be due to ruptured ectopic gestation. Patient stays still in the bed in supine position without moving in case of acute peritonitis as movements will increase the pain. Only in late stages when patient develops septic shock becomes restless irritable, grumbling. Patient rolls in the bed with agony in colicky pain. In initial period of acute peritonitis, acute appendicitis, and acute pancreatitis *pulse* remains normal but once sepsis develops and disease progresses tachycardia develops gradually. In ruptured ectopic there is only tachycardia to begin with. In acute intestinal obstruction, pulse becomes rapid due to dehydration. In acute abdomen initially *respiration* is normal but later tachypnoea may develop. That is due to septic shock or pleural effusion (in acute pancreatitis). *Fever* is a common feature of acute appendicitis or acute cholecystitis. Fever may not be present in many acute abdominal conditions. In some conditions like acute appendicitis fever suggests the severity of the disease. Once patient develops septicaemia (*cold shock*) fever may not be present. *Tongue* may be dry or coated. Brown tongue suggests toxemia. *Cyanosis* may be evident. *Pallor* and *jaundice* may be evident.

Urine output is important indicator in acute abdomen. 50 ml/hour urine is the required output. Hourly estimation is needed.

Local Examination of Abdomen

Inspection

Expose the abdomen from nipples to middle of the thighs for proper inspection.

Movements with Respiration

It should be observed by the side of the patient with eyes at the level of the abdomen. Restricted movement of the abdomen with respiration is seen in peritonitis. All quadrants show restrictions. Localized limitation of the movement with respiration is seen in localized diseases like appendicitis or cholecystitis.

Contour of the Abdomen

Distension of the abdomen is gradually progressive in acute intestinal obstruction. It is minimal or absent in proximal small bowel obstruction; it is central in distal small bowel obstruction in umbilical region; it is peripheral in large bowel obstruction. It is not obvious in acute appendicitis, acute cholecystitis. Distension is gradually progressive in acute peritonitis. It is not observed and abdomen remains scaphoid in biliary or ureteric colic. Abdominal girth should be measured in acute abdomen cases (at the level of umbilicus) and should be repeated at regular intervals (2nd hourly) to check the progression. Flank fullness should be observed (**Fig. 24.3**).

Visible Peristalsis

Step ladder peristalsis is typical of small bowel obstruction. It suggests intestinal obstruction (**Fig. 24.4**).

Skin over the Abdomen

Flank discolouration (*Grey Turner's sign*) and discolouration around umbilicus (*Cullen's sign*) may be a feature of acute haemorrhagic pancreatitis. Skin stretch, oedema of skin are other features to be observed. Umbilicus may be everted in abdominal distension. Old scar of laparotomy if present should be inspected in detail.



Fig. 24.3: Distension of abdomen due to dilated colon—could be due to sigmoid volvulus.



Fig. 24.4: Step ladder peristalsis of small bowel obstruction.

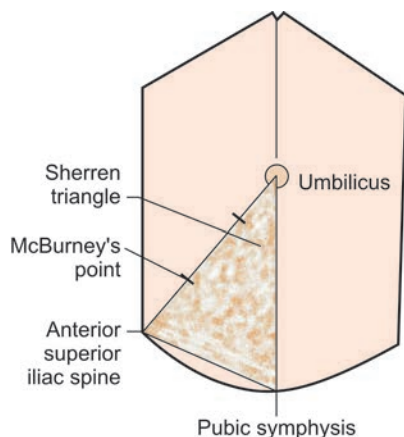
Hernial orifices should be inspected for impulse on coughing. One of the common causes of intestinal obstruction is obstructed inguinal hernia. Impulse on coughing may be absent in obstructed hernia but swelling will be obvious. Even femoral, umbilical or incisional hernia can cause intestinal obstruction.

Palpation

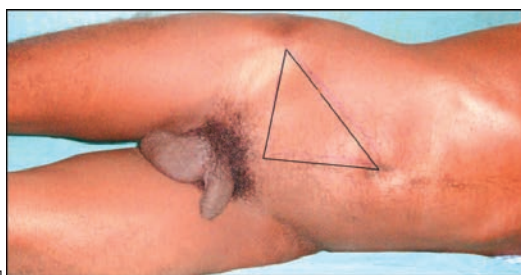
Examiner should keep his *hands warm*. Fingers should be kept flat over the abdomen to palpate from ventral surface of the fingers not from the tip of fingers.

Hyperaesthesia

Inflamed abdominal organ causes cutaneous hyperaesthesia which can be confirmed by scratching the skin or gently holding the fold of skin. *Sherren's triangle* is formed by lines joining umbilicus, anterior superior iliac spine and pubic symphysis which show hyperaesthesia in acute gangrenous appendicitis. Once this appendix bursts hyperaesthesia disappears. In acute cholecystitis an area of hyperesthesia is often evident between 9th to 11th ribs behind on right side – *Boas's sign* (Figs 24.5A and B).



A



B

Figs 24.5A and B: Sherren's triangle – hyperaesthesia zone. Also note the McBurney's point.

Tenderness

Point tenderness is elicited using one finger while palpating directly over the site of the organ. *McBurney's* tenderness is typical of acute appendicitis which is of spinoumbilical line at junction of medial 2/3rd and lateral 1/3rd. Tenderness in acute cholecystitis is located in the tip of 9th costal cartilage near the lateral margin of the right rectus muscle. Tender area will

be more clear if patient coughs. Extent and severity of tenderness should be elicited. Tenderness can also be elicited by percussion over the location. Appendicular tenderness will be better elicited in lateral position as intestines will be shifted towards left side and abdomen will be relaxed (**Fig. 24.6**).



Fig. 24.6: Palpation of abdomen using flat of the fingers gently. First tenderness is elicited.

Rebound Tenderness / Release Sign / Blumberg's Sign

Abdomen is gently palpated with every expiration reaching depth. When the hand is abruptly removed to spring back the abdominal muscles to its original position, patient winces with pain. It is due to inflamed parietal peritoneum in acute peritonitis which also springs back along with muscles. It is useful in acute appendicitis, acute peritonitis, and intestinal obstruction with bowel becoming gangrenous (**Fig. 24.7**).

Rovsing's Sign

When pressed in the left iliac fossa intestine gets pushed towards right iliac fossa pressing the inflamed appendix causing tenderness in right iliac fossa which is a definitive feature of acute appendicitis (**Fig. 24.8**).

Cope's Psoas Test

Retrocaecal appendicitis irritates psoas major muscle causing flexion of right hip joint. Patient develops



Fig. 24.7: Rebound tenderness.



Fig. 24.8: Rovsing's sign.

pain when this muscle is stretched by hyperextending the hip joint with patient turning towards his left side (**Fig. 24.9**).

Cope's Obturator Test

Stretching of the obturator internus muscle by internally rotating the hip joint will cause pain if inflamed appendix is in pelvic position and gets irritated by the obturator internus muscle (**Fig. 24.10**).

Baldwing's Test/Sign

Flank is pressed with hand and with knee extended patient raises his right hip from the bed to cause



Fig. 24.9: Cope's psoas test.



Fig. 24.10: Cope's obturator test.

contraction of psoas major muscle which irritates inflamed retrocaecal appendix to develop pain.

Muscle Guarding and Rigidity

It is *very important sign* of parietal peritonitis. It is involuntary abdominal wall muscle contraction which is present both during inspiration and expiration. Patient is asked to take deep slow breathing through his mouth. With fingers kept flat over the abdomen rigidity is checked in both phases of respiration. Voluntary guarding brought by the patient due to fear will disappear during expiration. Guarding is checked all over the abdomen. It is also checked using both hands kept one over the other. Deeper hand is passively feeling the rigidity; outer hand presses the abdomen through deeper hand. Localized rigidity is often observed in different conditions like in right upper abdomen in case of perforated duodenal ulcer in initial period; in right iliac fossa in acute appendicitis; in the loin, in retrocaecal appendicitis. Guarding is absent in pelvic appendicitis, ureteric/biliary colic, in early acute intestinal obstruction without bowel gangrene (**Fig. 24.11**).



Fig. 24.11: Both hands are used to palpate to overcome the rigidity.

Mass Abdomen

Detailed features of the mass should be assessed – examples like appendicular mass, colonic mass.

Other Palpation

Palpation for distension of abdomen, hernial sites are important.

Percussion

Tenderness may be elicited by percussion. Free fluid can be checked by same methods like shifting dullness, fluid thrill. It should be gentle as pain will be more. Just percussion in the flanks to elicit dullness may be sufficient if patient is in agony.

Obliteration of liver dullness should be specifically checked in acute abdomen. It is the definitive sign of bowel perforation. Upper border of liver is checked by percussing in right midaxillary line from above downwards. Obliteration of liver dullness with resonant note throughout is due to presence of gas/air between diaphragm and liver. It is present in 70% of perforations. Gas under diaphragm may not be evident if gas leak is less; if patient has undergone laparotomy earlier causing adhesions above liver, if perforation is in ileum. Interposition of colon/intestines between liver and diaphragm causes obliteration of liver dullness.

Auscultation

Absence of bowel sounds causing *silent abdomen* is typical pathognomonic sign of diffuse peritonitis.



Fig. 24.12: Auscultation of the abdomen for bowel sounds is important. Silent abdomen is a feature of acute peritonitis. In early intestinal obstruction bowel sounds are increased.

Bowel sounds are increased in early phase of intestinal obstruction with metallic tinkles or borborygmi. Normal bowel sounds are called as 'clicks or gurgles' (**Fig. 24.12**).

Digital Examination of the Rectum

Ballooning of rectum which is empty is a feature of acute intestinal obstruction or acute peritonitis. It is probably of neuronal origin but exact cause is not clear. Tenderness over the right side is felt in acute appendicitis. Tenderness in retrovesical/retrouterine pouch is a feature of diffuse peritonitis or pelvic peritonitis. Pelvic abscess often may be felt in front as a boggy swelling which is soft, and tender. Rectal examination is done in left lateral position usually but dorsal position may be used if patient is severely ill. Red currant jelly in the stool may be seen in intussusception.

Pervaginal Examination

It is important in acute salpingitis, ruptured ectopic gestation, twisted ovarian cyst, etc.

Examination of External Genitalia

Scrotum, testes, cord and vas deferens should be examined as inflammation in these organs mimics acute abdominal condition especially acute appendicitis and ureteric colic.

Systemic Examination

Examination of respiratory and cardiac system is done to look for pneumonia, pleurisy, basal pneumonia, myocardial infarction or angina pectoris. All these conditions are known to present as pain abdomen but abdomen will be soft without distension and other features of specific diseases are evident.

Herpes zoster infection may present like acute abdomen pain (acute cholecystitis) due to hyperesthesia.

Tuberculosis of spine (Pott's) may present like acute abdomen in children and so spine should be examined (Fig. 24.13).

Neurological examination (Central and peripheral nervous system).



Fig. 24.13: Spine should be examined in acute abdomen cases.

Many neurological conditions mimic acute abdomen. Tabes dorsalis with gastric crisis is a known entity presenting as acute pain in the abdomen and vomiting. Lightning pain in legs, papillary changes (Argyll Robertson), absence of knee and ankle jerks are obvious in tabes dorsalis.

Investigations

1. **Blood:** Leucocytosis is common in peritonitis. In septicaemia count may be decreasing so serial estimation is needed.

Blood urea, serum creatinine and electrolytes are estimated to confirm uraemia and electrolyte imbalance due to dehydration or sepsis.

Liver function tests, platelet count, prothrombin time estimation, serum calcium, amylase, lipase are done in acute pancreatitis.

Plasma fibrinogen, C reactive protein (normal is 6 mg/L), serum methaemoglobin are often specific in inflammatory conditions.

Urinary lipase, diastase (normal is 10-30 units) are relevant in acute pancreatitis. Urine analysis is a must for pus cells and red cells.

2. **Plain X-ray abdomen** is simple and very relevant investigation.

It is taken in erect posture or lateral decubitus position in severely ill patients. Gas under diaphragm (perforation), multiple air fluid levels of intestinal obstruction, calcified areas in the pancreatitis (stones) are looked for. Jejunum shows volvulae conniventis; ileum is characterless; colon shows haustrations. More the number of air fluid levels distal is the obstruction. Normal X-ray shows 2 or 3 air fluid levels—fundus, duodenum, occasionally caecum. Ground glass appearance is a feature of acute peritonitis (Figs 24.14 to 24.16).

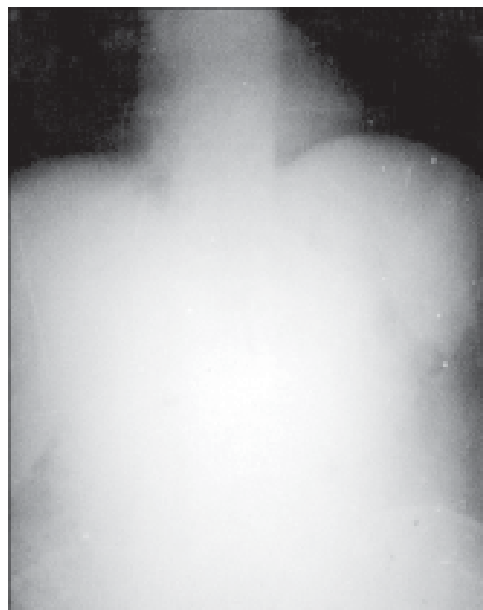


Fig. 24.14: Plain X-ray abdomen showing ground glass appearance—a feature of acute peritonitis.



Fig. 24.15: Plain X-ray abdomen showing multiple air fluid levels—a feature of intestinal obstruction.



Fig. 24.16: X-ray abdomen in erect posture showing gas under diaphragm. In severely ill patient, X-ray lateral decubitus is taken.

3. **US abdomen** will show air in the peritoneal cavity. Poor window due to dilated bowel makes it difficult to identify the cause.
4. **CT abdomen** is very relevant investigation in intestinal obstruction, acute peritonitis and acute pancreatitis. In intestinal obstruction CT is useful but contrast into the bowel (orally) cannot be given (IV contrast can be given).

5. **Cholescintigraphy** using HIDA – hippuric iminodiacetic acid or I^{131} Rose Bengal radioisotope scan has got 100% accuracy in diagnosing acute cholecystitis.

Laparoscopy is newer modality to identify the pathology. If perforation is identified it can be closed through laparoscopy.

Note: Gastrointestinal contrast studies (barium) or endoscopy is not done in acute abdomen. Occasionally dilute gastrograffin contrast study can be done with care. Barium will leak into the peritoneal cavity causing chemical peritonitis.

Acute Appendicitis

Etiology

(1) Altered diet. (2) Familial susceptibility. (3) Obstruction of the lumen of appendix causing obstructive appendicitis. *Blockage* may be due to Faecoliths, stricture, foreign body, roundworm or threadworm. *Adhesions and kinking*—Carcinoma caecum near the base, ileo-caecal Crohn's disease. (4) Distal colonic obstruction. (5) Abuse of purgatives. Faecolith is the commonest cause.

Organisms

E. coli (85%), Enterococci (30%), Streptococci, anaerobic streptococci, *Cl. welchii*, *Bacteroides*.

Types

1. **Acute non-obstructive appendicitis:** Inflammation of mucous membrane with redness, oedema and haemorrhages which may go for following courses: Resolution; Ulceration; Fibrosis; Suppuration; Recurrent appendicitis; Gangrene; Peritonitis.
2. **Acute obstructive appendicitis:** Here pus collects in the blocked lumen of the appendix which has become blackish, gangrenous, oedematous and rapidly progresses leading to perforation either at the tip or at the base of the appendix (**Fig. 24.17**). This will lead to peritonitis, formation of appendicular abscess or pelvic abscess. Most often there will be thrombosis of the appendicular artery.
3. **Recurrent appendicitis:** Repeated attacks of non obstructive appendicitis leads to fibrosis, adhesions causing recurrent appendicitis.



Fig. 24.17: Gangrenous appendicitis.

4. *Subacute appendicitis* is milder form of acute appendicitis.

Clinical Features

1. It is rare before the age of two, common in children and other age groups.
2. **Pain:** Visceral pain starts around the umbilicus due to distension of the appendix, later after few hours somatic pain occurs in right iliac fossa due to irritation of parietal peritoneum by the inflamed appendix. Pain eventually becomes severe and diffuse which signifies spread of infection into the general peritoneal cavity.
3. **Vomiting:** Due to reflex pylorospasm.
4. **Constipation** is the usual feature *but diarrhoea* can occur if appendix is in post-ileal or pelvic positions.
5. **Fever, tachycardia, foetor oris** are other features.
6. **Urinary frequency:** Inflamed appendix may come in contact with bladder and can cause bladder irritation.
7. Tenderness and rebound tenderness in right iliac fossa (*release sign—Blumberg's sign*) are typical.
8. **Rovsing's sign:** On pressing left iliac fossa, pain occurs in right iliac fossa which is due to shift of

bowel loops which irritates the parietal peritoneum.

9. Hyperextension (in case of retrocaecal appendix—*Cope's psoas test*) or internal rotation (in case of pelvic appendix—*obturator test*) of right hip causes pain in right iliac fossa due to irritation of psoas muscle and obturator internus muscle respectively.
10. **Baldwing's test** is positive in retrocaecal appendix—when legs lifted off the bed with knee extended, the patient complains of pain while pressing over the flanks.
11. P/R examination shows tenderness in right side of the rectum. Often infection gets localized by omentum, dilated ileum and parietal peritoneum leading to *appendicular mass*. Most often sup-puration occurs in the localized area resulting in *appendicular abscess*.

Clinical signs in appendicitis

1. Rovsing's sign
2. Blumberg's sign (release sign)
3. Cope's psoas test
4. Obturator test
5. Baldwin's test

Acute Appendicitis in Infancy

Eventhough it is rare, when it occurs, it has got 80% chances of perforation with high mortality (50%).

Acute Appendicitis in Children

Here localization is not present, and so peritonitis occurs early. It *requires early surgery*. Dehydration, septicaemia is common.

In Elderly

Gangrene and perforation are common. Because of lax abdominal wall, localisation is poor and so peritonitis sets in early.

In Pregnancy

Appendix shifts to upper abdomen. So pain is higher and more lateral. After 6 months, maternal mortality increases by 10 times than usual and also leads to premature labour.

Differential diagnosis

Perforated peptic ulcer.	Ruptured or twisted ovarian cyst.
Acute cholecystitis.	Right ureteric colic.
Enterocolitis.	Right acute pyelonephritis.
Mesenteric lymphadenitis.	Lobar pneumonia.
Crohn's disease.	Acute pancreatitis.
Meckel's diverticulitis.	Acute crisis of porphyria.
Salpingitis.	Diabetic abdomen, etc.
Ectopic gestation-ruptured.	

Investigations

Total leucocyte count is increased. US is done to rule out other conditions like ureteric stone, pancreatitis, ovarian cyst, ectopic pregnancy and also to confirm appendicular mass or abscess.

Alvarado scoring for appendicitis:**Score:**

Migrating pain.	1
Anorexia.	1
Nausea and vomiting.	1
Tenderness in right iliac fossa.	2
Rebound tenderness.	1
Elevated temperature.	1
Leucocytosis with count more than 10,000.	2
Shift to left with neutrophilia in peripheral smear	1
Total score:	10

Score less than 5: Not sure.

Score between 5-6: Compatible.

Score between 6-9: Probable.

Score more than 9: Confirmed.

Acute Cholecystitis

Commonly it occurs in a patient with pre-existing chronic cholecystitis but often also can occur as a first presentation. Usual cause is impacted gallstone in the Hartmann's pouch obstructing cystic duct.

Bacteria are

E. coli
Klebsiella
Strep. faecalis
Salmonella
Clostridium welchii.

Clostridium welchii can cause gas in the gallbladder and also septicaemia which can be life threatening-*emphysematous cholecystitis*.

Classification

Acute calculous cholecystitis and *acute acalculous cholecystitis*. Acute cholecystitis can lead to *perforation*, which usually occurs in the fundus or in the neck (Hartmann's).

It can cause cholecystoduodenal, cholecysto-intestinal or cholecystobiliary fistula, peritonitis, pericholecystic abscess, cholangitis and septicaemia.

Clinical Features

Sudden onset of pain in the right hypochondrium, with tenderness, guarding, and rigidity. Palpable, tender, smooth, soft gallbladder is evident. Area of hyperaesthesia between 9th and 11th ribs posteriorly on the right side (*Boas's sign*). Jaundice may be present.

Differential Diagnosis:

Duodenal ulcer perforation
 Acute pancreatitis
 Acute appendicitis
 Acute pyelonephritis
 Lobar pneumonia
 Ruptured ectopic pregnancy.

Investigations:

US abdomen
 Plain X-ray abdomen
 Total count (neutrophilia)
 HIDA radio-isotope study (very useful).

Acute Pancreatitis

Trapnell's aetiological classification:

Major causes.

Biliary tract disease 50%
 Alcoholism 25%

Other causes:

Trauma
 After biliary, gastric, splenic surgery
 Hyperparathyroidism
 Hypercalcaemia
 Diabetes
 Porphyria
 Drugs: Steroids, INH, diuretics, septran, azathioprine
 Viral infections (mumps, coxsackie)
 Autoimmune diseases
 Vascular diseases
 Idiopathic.

Pathogenesis

All these etiological factors → Either cause the spasm of sphincter of Oddi and lead to reflux of bile into pancreatic duct and into the pancreatic parenchyma or cause increased secretion of pancreatic enzymes or directly act as toxins to pancreas. Trypsinogen gets activated forming trypsin which activates other enzymes. Proelastase to elastase → causes capillary rupture; Prolipase to lipase → metabolises triglycerides to glycerol + fatty acids and fatty acids combine with

calcium forming saponified fat. Sequestered fluid, saponified fat, blood, toxins all together forms a *chicken broth fluid*. Lecithinase, amylase and other proteolytic enzymes are also released. Infection occurs causing bacteraemia, septicaemia. Large volume of fluid sequestration causes hypovolaemic shock. Toxins released may lead to acute tubular necrosis and so acute renal failure. Left sided diaphragm gets elevated and so left sided pleural effusion occurs. Lecithinase reduces the surfactant in the alveoli of lung, and infection leads to pulmonary insufficiency, ARDS and respiratory failure. Because calcium is utilised for saponification, *hypocalcaemia* sets in. Diffuse oozing in pancreatic bed occurs which utilises platelets and causes *disseminated intravascular coagulation (DIC)*. In severe cases, extensive necrosis with haemorrhage occurs causing acute *haemorrhagic necrotising pancreatitis (Fulminant pancreatitis)*, which has got a high mortality. Here enzymes seep across the retroperitoneum causing haemorrhagic spots and ecchymosis in the flanks (*Grey Turner's sign*), or through falciform ligament causing discolouration around the umbilicus (*Cullen's sign*), or below the inguinal ligament (*Fox sign*).

Moynihan described acute pancreatitis as *the most terrible of all the calamities that occur in connection with abdominal viscera*.

Clinical Features

Presents with sudden onset of upper abdominal pain which is referred to back, vomiting, high fever, often mild jaundice (due to cholangitis). Clinical signs include tachypnoea with cyanosis, tenderness, rebound tenderness, guarding, rigidity and abdominal distension, features of shock and dehydration, oliguria. Grey turner's sign, Cullen's sign, Fox sign.

Differential Diagnosis

Perforated duodenal ulcer
Cholecystitis
Mesenteric ischaemia
Ruptured aortic aneurysm
Ectopic pregnancy
Salpingitis
Intestinal obstruction
Diabetic ketoacidosis.

Investigations

Serum amylase is very high (>1000 Somogyi units) or will show rising titre. Serum lipase more specific than amylase. Serum lactescence (related to triglyceride metabolism). Liver function tests: Serum bilirubin, albumin, prothrombin time, alkaline phosphatase; Blood urea, serum creatinine. Blood glucose (hyperglycaemia is seen). Serum calcium level, (hypocalcaemia occurs). Arterial PO₂ and PCO₂ level to assess pulmonary insufficiency (or ARDS). Urinary lipase estimation. Total count, haematocrit. Peritoneal tap fluid shows *high amylase* and protein level (very useful method). Plain-X-ray shows: (1) *sentinel loop* of dilated proximal small bowel, (2) distension of transverse colon with collapse of descending colon (*colon cut off sign*), (3) Air-fluid level in the duodenum. US abdomen; Spiral CT; MRCP; ERCP is usually not done in acute phase.

Acute Peritonitis

Types

Primary; Secondary; Tertiary.

Primary peritonitis is commonly due to *Pneumococcus* and seen in young girls between 3 and 6 years. Infection spreads from lower genitals through fallopian tubes, from upper respiratory tract infection or from middle ear in males. It is uncommon after 10 years of age. It is common in malnourished child and child with nephritis. Child is toxic, severely ill and will go into septicaemia very early. TC is very high >30,000.

Secondary peritonitis is secondary to any bowel or other visceral pathology, e.g. Perforation, appendicitis.

Tertiary peritonitis occurs after the treatment for any abdominal surgeries, which is usually severe and may go in for SIRS or MODS early. *Mortality for diffuse peritonitis is 10%.*

- a. *Primary*: Mono-microbial, extraperitoneal source
- b. *Secondary*: Most common, polymicrobial, intraperitoneal source
- c. *Tertiary*: Due to superadded infection following treatment of secondary peritonitis

Bacteria Causing Peritonitis

(a) *Bacteria from GIT*—*E.coli*, anaerobic streptococci, anaerobes (bacteroides), *Klebsiella*, *Cl. welchii*.
 (b) *Bacteria not from GIT*—Gonococcus, pneumococcus, streptococcus are from fallopian tubes—occurs in young females, commonly. Most common bacteria during peritonitis phase is *E.coli* and during abscess formation is *B. fragilis*.

Mode of Infection

Perforation of the GIT; penetrating or blunt trauma; surgery; drains; dialysis; foreign body; appendicitis, cholecystitis; intestinal obstruction with strangulation; via fallopian tubes; through blood spread.

Factors Affecting the Spread of the Infection in Peritonitis

Rapidity by which the pus is gushed into the peritoneal cavity (e.g. burst appendix, perforations). Amount of peristalsis (more the peristalsis more the spread); Virulence of the organism; Localising action of the omentum (in children localisation is poor as omentum is small); Immunosuppression; Anatomical nature of the peritoneal cavity, etc.

Clinical Features

Sudden onset of pain; fever, vomiting, tenderness initially localised later becomes diffuse; rebound tenderness; guarding and rigidity; tachycardia, tachypnoea; tenderness on P/R examination; distension with silent abdomen; eventually leading to, septicaemic shock, *Hippocrates facies* and loss of consciousness.

Investigation

Plain-X-ray abdomen—will show *ground-glass appearance* with gas under diaphragm in the presence of perforation; Total count will be increased; US abdomen to show fluid in the abdominal cavity; electrolyte study; blood urea and serum creatinine.

Differential Diagnosis

Pancreatitis; intestinal obstruction; ruptured ectopic pregnancy; pyelonephritis; acute mesenteric ischaemia; diabetic acute abdomen.

Subphrenic Spaces and Subphrenic Abscess

There are four intra peritoneal and three extra-peritoneal spaces.

Intraperitoneal Spaces

Right Anterior Intraperitoneal Space

It is bounded by right lobe of the liver and diaphragm, coronary and right triangular ligament, and to the left by falciform ligament.

Causes: Abscess here occurs due to cholecystitis, perforated duodenal ulcer, postoperative, appendicitis, duodenal cap blow out.

Right Posterior Intraperitoneal Space (Rutherford Morrison's Kidney Pouch)

It is bounded in front by the liver and gallbladder, above by the liver, behind by the right, kidney and diaphragm, below by the transverse colon and hepatic flexure, to the left by foramen of Winslow and duodenum. It is large and deepest space of all. *It is the commonest site for subphrenic abscess.*

Causes: Appendicitis, cholecystitis, postoperative, perforated duodenal ulcer, intestinal obstruction.

Left Anterior Intraperitoneal Space

It is bounded above by the diaphragm, behind by left lobe of liver and left triangular ligament, gastrohepatic ligament and anterior surface of the stomach, to the right is the falciform ligament.

Causes for abscess here are surgeries of the stomach, tail of the pancreas, spleen, colon (splenic flexure), diverticulitis.

Left Posterior Intraperitoneal Space

It is bounded by stomach, pancreas, greater omentum, liver, transverse colon (Lesser sac). Commonest cause here is pseudo-cyst of pancreas. Rarely perforated gastric ulcer.

Extraperitoneal Spaces

Right extraperitoneal space is right perinephric space and *left extraperitoneal space* is left perinephric space.

Causes: Abscess here are due to tuberculosis, trauma, haematoma.

Midline extraperitoneal space is bare area of the liver. Pus collects here commonly due to amoebic liver abscess and pyogenic abscess of the liver.

Clinical Features

Pus somewhere, pus nowhere else, pus under diaphragm—Bernard's aphorism. History relevant to the specific causes, H/O previous surgery. Fever with chills and rigors; Pain in right hypochondrium; Tenderness in right hypochondrium; Sympathetic right sided pleural effusion due to congestion and hyperaemia of the diaphragm; Pain in the right shoulder due to irritation of phrenic nerve; Hiccough, tachycardia, tachypnoea.

Differential Diagnosis

Amoebic abscess; Pylephlebitis; Empyema; Pulmonary collapse.

Investigations

Plain X-ray chest and abdomen shows soft tissue shadow, pleural effusion, tenting of diaphragm, collapse of the lung. US abdomen confirms the diagnosis. TC is high. CT chest is diagnostic (**Fig. 24.18**).



Fig. 24.18: CT scan showing left subphrenic abscess.

Complications

Empyema; respiratory arrest; septicaemia.

Amoebic Liver Abscess

It is common in India and other tropical countries and it is caused by a parasite *Entamoeba histolytica*. It is more common in *alcoholics and cirrhotic* patients.

Pathology

Initially from infected recto-sigmoid or ileo-caecal region, amoebic trophozoites reach the liver through portal veins causing amoebic hepatitis may be in the form of micro-abscesses all over the liver. This might resolve on its own or with antiamoebic drugs, but many times lead to a localised *amoebic liver abscess*. In 70% of cases it is *single large abscess*, in 30% it is multiple, that may involve both lobes. Problems and difficulties in treating, in addition to poor prognosis is more common in *multiple abscesses*. Amoebic liver abscess is more common in *right posterior-superior* region because of streamline effect, i.e. the portal vein is in direct continuation with the right branch. It can be *multiloculated* also. Pus is *chocolate-coloured*, classically called as *anchovy-sauce*, contains dead liver cells, RBCs, necrotic material. Pus may be green due to bile admixture. Most often secondary infection by *E. coli*, *Staph.*, *Strepto.*, etc. may occur and so may present with features of pyogenic liver abscess. Because of perihepatitis, liver is fixed to diaphragm or abdominal wall. Hyperaemia in the diaphragm causes sympathetic *pleural effusion on right side*. Commonly amoebic abscess presents as an *acute* entity, but it can also present as *chronic type* which is covered by a capsule, that remains dormant for a long period. Sometimes it can get calcified also.

Course and Sequelae of Amoebic Liver Abscess

It can *rupture into lungs* leading to expectoration of chocolate-coloured sputum resulting in natural regression of abscess. It can rupture into the peritoneum causing *peritonitis* which requires emergency laparotomy. It can rupture into pleural cavity leading to *empyema* which is the commonest complication (**Fig. 24.19**). Rupture into bare-area causing *retro-peritoneal abscess*. Rupture into the intestines, or to the skin (*Amoebiasis cutis*). Most dangerous is rupture into pericardial cavity (*cardiac tamponade*) which has got very *high mortality* requiring emergency thoracotomy and pericardial decompression. *Septicaemia and liver failure* can occur in a patient with amoebic liver abscess with *cirrhosis*.

Clinical Features

It is common in males, may be after an attack of amoebic dysentery, or many months after the attack or history

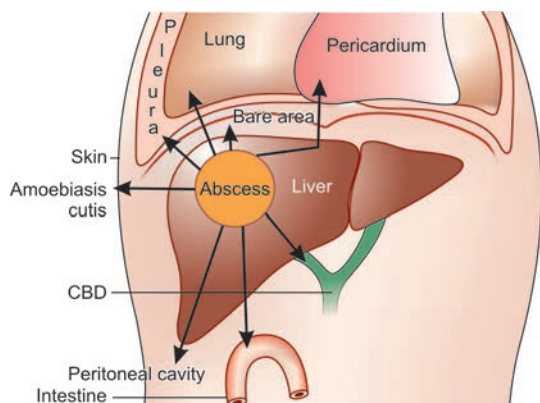


Fig. 24.19: Complications of amoebic liver abscess.

of dysentery may not be there at all. They present with fever, loss of weight, chills and rigors, pain in the right hypochondrium, soft, tender, smooth, liver with increased liver span. Intercostal tenderness is elicited which is a useful clinical sign. Right sided pleural effusion may be evident. Mild jaundice is not uncommon especially in cirrhotics and multiple abscesses which may signify poor prognosis. Tenderness, rigidity and skin oedema in right hypochondriac region may be present in acute cases. In chronic amoebic liver abscess, smooth, firm/hard, nontender liver may be palpable.

Differential Diagnosis

For Acute type: (1) Acute cholecystitis. (2) Acute presentation of hepato-cellular carcinoma (HCC) due to haemorrhage or necrosis. **Chronic abscess will mimic hepatoma in every respect.**

Perforated Duodenal Ulcer

It is common in males (8:1) between 35-45 years of age group but can occur in any age group. *Anterior ulcer* perforates. In 80% of cases there is a history of chronic DU. In 20 % cases it is silent perforation. Perforation can occur in acute ulcers or in acute presentation of a preexisting chronic ulcer. Perforation may be precipitated by steroids, analgesics (NSAIDs), alcohol, antimalarials. Overall incidence is 5% (**Fig. 24.20**).

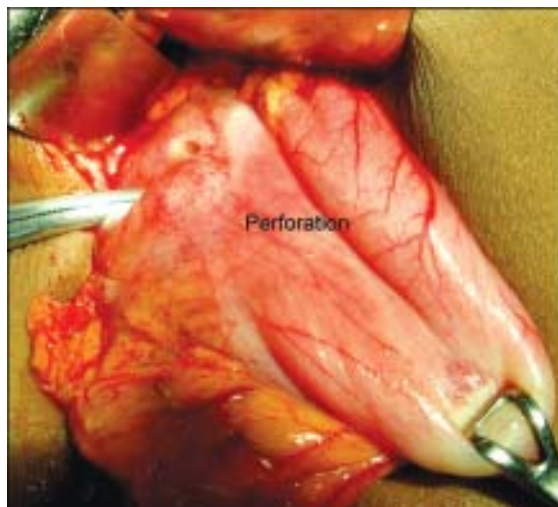


Fig. 24.20: Duodenal ulcer perforation.

Stages of perforation

Stage of chemical peritonitis

Once perforation occurs, stomach contents escapes into the peritoneal cavity. The acid from the stomach causes chemical peritonitis leading to severe pain in epigastric region, vomiting, tenderness, guarding, rigidity, tachycardia, sweating.

Stage of reaction (Stage of illusion)

Peritoneum secretes lot of fluid to neutralise the escaped content and so temporarily the pain reduces, and the patient feels better. It lasts for about 6 hours.

Stage of diffuse bacterial peritonitis

After about six hours, bacteria from GIT (escape) migrate from the site of perforation causing diffuse peritonitis.

Clinical Features

Patient is toxic, with tachycardia, hypotension, tachypnoea, vomiting. Presents with fever, dehydration, oliguria; tenderness and rebound tenderness all over the abdomen; guarding and rigidity, initially in the epigastrium but later all over the abdomen; dullness over the flank because of fluid; obliteration of liver dullness because escaped gas gets collected under the diaphragm; silent abdomen with absence of bowel sounds; tenderness felt on per rectal examination. Sometimes fluid from supracolic region slowly trickles down along the right paracolic gutter, collects in right iliac region causing pain and tenderness in right iliac fossa mimicking appendicitis. Often slow, small perforation presents with subacute features, but diffuse peritonitis sets in eventually in 24-48 hours.

Terminal Stage

Patient may have oliguria, septicaemia, shock, *Hippocratic facies* (sunken eyes, rapid breathing, ill look), with MODS (Multi organ dysfunction syndrome).

Investigations

Plain X-ray erect abdomen: Shows gas under diaphragm in 70% of cases. In 30% of cases, there will be no gas under diaphragm. It may be due to either the gas leak is less than 1 ml or due to previous surgery causing adhesions between liver and diaphragm.

Chilaiditi's syndrome is the interposition of the colon in front and above the liver. It is common in children and elderly. It may be mistaken for gas under diaphragm in plain X-ray abdomen. US abdomen shows free fluid and often gas.

Different signs in X-ray in perforation

Cupola sign—Crescent-shaped radiolucency under the diaphragm

Riglers sign—Visualisation of both aspects of the bowel wall being outlined by gas on either side

Inverted V sign—Gas on either sides of the falciform ligament

Football sign—Collection of gas in the centre of the abdomen like a foot ball

Triangle sign—Gas between bowel loops

Conditions which mimic pneumoperitoneum- pseudo-pneumoperitoneum

Subpulmonary pneumothorax

Chilaiditi syndrome

Subphrenic abscess due to infections by gas forming organism like *Clostridium welchii*

Subdiaphragmatic fat or omental fat under the diaphragm may rarely mimic gas under the diaphragm

Perforation of Typhoid Ulcer

Perforation usually occurs in 3rd week of the infection. Ulcers are multiple, arranged in parallel and in anti-mesenteric border of the ileum. One or more ulcers may perforate and many ulcers may be on impending perforation. Patient is toxic presents with, severe diarrhoea, relative bradycardia, soft abdomen, obliterated liver dullness, abdomen without guarding and rigidity (because of Zenker's degeneration). Possibility of missing typhoid perforation is very high. X-ray in erect posture will show gas under diaphragm. Widal test is positive. Blood culture and stool cultures are often required as other methods for diagnosis.

Meckel's Diverticulum

It is 2% common; 2 feet from the ileo-caecal valve; 2 inch in length. It is congenital, results from incomplete closure of vitello-intestinal duct. It arises from the anti-mesenteric border of the ileum, contains all three layers of the bowel with independent blood supply. In 20% of cases mucosa contains heterotopic epithelium like gastric, colonic and or pancreatic tissues. It may be connected to or communicate with the umbilicus through a band or fistula.

Presentations

Severe haemorrhage—most common, seen in children aged 2 yrs or younger; intestinal obstruction, perforation, intussusception, peptic ulceration, diverticulitis—features will mimic acute appendicitis; Littre's hernia; silent Meckel's diverticulum found during laparotomy or laparoscopy or by radioisotope study

Diagnosis

Technetium Tc⁹⁹ radioisotope scan is very useful. X-ray abdomen to look for complications like obstruction, perforation, etc. *Laparoscopy is very useful.*

Intestinal Obstruction

Classification: Congenital, Acquired.

Congenital	Acquired
Anorectal malformations	Hernia (commonest)
Congenital megacolon	Postoperative
Adhesions	Intussusception
Duodenal atresia	Roundworm
Intestinal atresia (ileal) (Fig. 24.21)	Gallstones
Bands and adhesions	Tuberculosis
Malrotation (Fig. 24.22)	Malignancy
Volvulus neonatorum	Internal hernias

Pathology

Changes proximal to the bowel obstruction: Intestinal obstruction → Increased peristalsis → Becomes vigorous → Obstruction not relieved → Peristalsis ceases → Flaccid paralysed dilated bowel. Fluid collects just proximal to the obstruction which is derived from saliva, stomach, pancreas and intestine. Because of oedema and inflammation absorption decreases, sequestration of fluid from the circulation into the lumen occurs and bacteria (*E. coli*, *Klebsiella*,

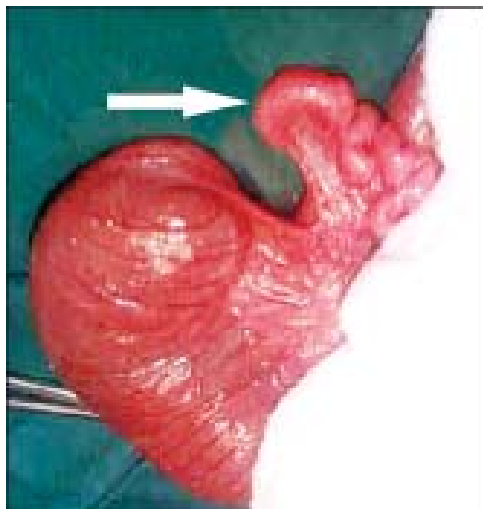


Fig. 24.21: Intestinal atresia.



Fig. 24.22: Malrotation on table look – is one of the causes of intestinal obstruction.

anaerobes, bacteroids and other organisms) multiply, toxins are released—*toxaemia* occurs. This leads to severe dehydration, electrolyte imbalance. Proximal to the collected fluid, air accumulates [derived from swallowed air (70%), diffusion from blood into the lumen (20%), from digested product and bacterial action (10%)], in which main component is nitrogen (90%) and also hydrogen sulphide. During vigorous peristalsis air enters the distal fluid, results in *churning*, is the reason to cause *multiple air-fluid levels in plain X-ray abdomen*.

Changes at the site of the obstruction: Initially venous return is impaired → Congestion, oedema of bowel wall which turns purple → later this jeopardises the arterial supply → Loss of shininess, black colour, loss of peristalsis → *Gangrene and perforation* occurs → Bacteria and toxins migrate into the peritoneum → *peritonitis*.

Closed Loop Obstruction

When there is obstruction in the large bowel, with ileo-caecal valve competence, pressure increases in the caecum → Stercoral ulcer form in the caecum → Gangrene → Perforation → Peritonitis (Faecal). Perforation also can occur at the site of obstruction due to the malignant growth (**Fig. 24.23**).

Bowel distal to the obstruction is inactive and collapsed.

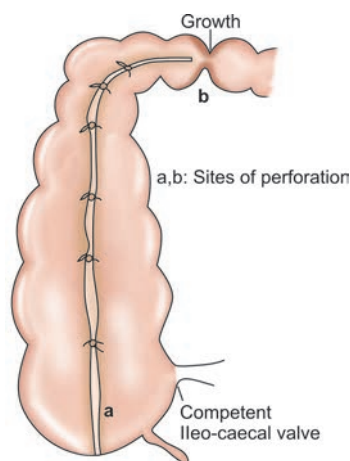


Fig. 24.23: Closed loop obstruction.

Clinical Features

Abdominal pain: Initially colicky and intermittent; later continuous and severe.

Vomiting: In *jejunal* obstruction it is early and persistent. In *ileal* obstruction, it is recurrent occurring at an interval; initially bilious later faeculent. In *large bowel* obstruction, vomiting is a late feature.

Distension: It is absent or minimal in case of *jejunal* obstruction; obvious with visible intestinal peristalsis (VIP) and borborygmi sounds in case of ileal

obstruction. It is enormous in case of large bowel obstruction.

Constipation: It is *absolute*, i.e. neither faeces nor flatus is passed. *Exceptions* where constipation may not be there Richter's hernia obstruction; Gallstone obstruction; Mesenteric vascular occlusion; Intestinal obstruction with a pelvic abscess.

Dehydration → Oliguria → Renal failure.

Features of toxæmia and septicaemia: Tachycardia, tachypnoea, fever, sunken eyes, cold periphery.

Features of strangulation: Shock, tenderness, rebound tenderness, guarding and rigidity, absence of bowel sounds. In case of strangulated hernia, a swelling which is tense, tender, rigid, irreducible, no expansile impulse on coughing and H/O recent increase in size is seen.

Per-rectal examination: Shows *empty, dilated rectum*, often with tenderness. If rectal growth is the cause for obstruction, it may be palpable.

Investigations

Plain X-ray abdomen: Multiple air-fluid levels. Proximal the obstruction lesser is the air fluid level; distal the obstruction more is the fluid level. Normally, three fluid levels can be seen in plain X-ray film—at fundus of stomach, at duodenum and often at caecum. *Jejunum* shows concertina effect due to *valvulae conniventes*. *Ileum* is smooth and characterless. Large bowel shows *haustration*.

Barium enema and meal is contraindicated in acute intestinal obstruction (**Fig. 24.24**).

Adhesions and Bands

Adhesions and bands are the commonest cause of intestinal obstruction in western countries. In India hernia and then adhesions are the two common causes of intestinal obstruction.

Causes

Infection due to peritonitis, appendicitis, post laparotomy, and other acute infective abdominal conditions; Materials used during surgery can cause dense inflammatory reactions—suture materials like silk, thread, and foreign body, mop, and gauze, talc powder; drugs like sulphonamides and penicillins; Ischaemia

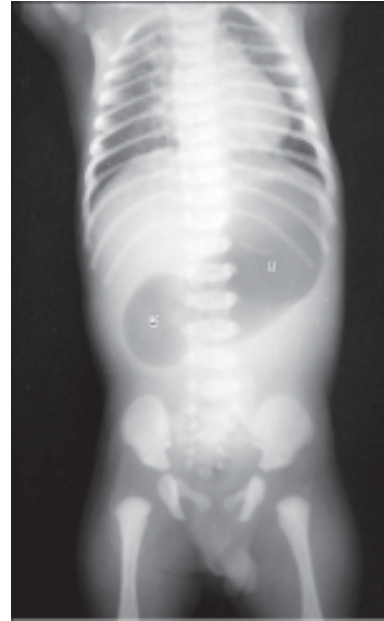


Fig. 24.24: Double bubble sign—feature of duodenal atresia.

of bowel due to poor blood supply, sepsis; Gynaecological conditions, bowel injury, radiation-induced enteritis, Crohn's disease, other inflammatory bowel diseases; Specific conditions like tuberculosis, malignancy.

Types

Type I—Fibrinous adhesions occur during 5–10th post-surgical period. It usually gets resolved completely. It is avascular and flimsy. **Type II—Fibrous** adhesions. Due to lack/poor blood supply bowel gets attached to part of peritoneum or omentum or other part of the bowel with dense vascular adhesions to maintain blood supply. It will persist and precipitate intestinal obstruction, often-subacute and recurrent type. *Adhesions due to tuberculosis are severe, dense and difficult to separate.*

Clinical Features

Pain abdomen—colicky type: recurrent and episodic. Distension, vomiting; constipation; reduced bowel sounds on auscultation; previous surgical scars commonly observed; dehydration, tachycardia, hypotension are other features (**Figs 24.25 to 24.27**).

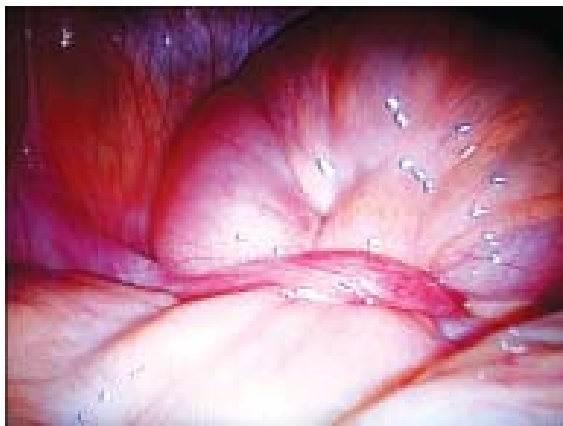


Fig. 24.25: Laparoscopic view of band in the right iliac fossa causing intestinal obstruction.



Fig. 24.27: Postoperative adhesive obstruction causing lower abdomen distension.

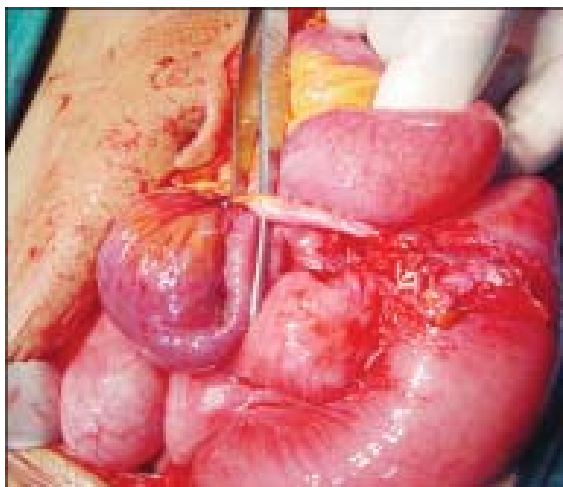


Fig. 24.26: Open surgery showing postoperative adhesive band causing intestinal obstruction.

does not move with respiration, resonant or impaired resonant on percussion are the features. It is often recurrent. Empty right iliac fossa is observed (Sign de Dance). *Red currant jelly stool* is common. Microbarium enema X-ray shows *claw sign* (Figs 24.28 and 24.29). US is diagnostic.



Fig. 24.28: Barium enema contrast X-ray showing claw sign – feature of intussusception. Nowadays microbarium sulphate solution is used. Air is also used commonly.

Acute Intussusception

It is telescoping of one segment of bowel to adjacent bowel. Ileocolic is the commonest type. It occurs in infants between 6-12 months. During weaning period Peyer's patches of the ileum gets hypertrophied and act as point for intussusception. Polyp, submucosal tumours are other causes. Pain, distension, features of obstruction, constipation are the feature. A sausage shaped mass is palpable in the abdomen usually towards left side with concavity towards umbilicus which is smooth, firm, contracts under palpating fingers, intermittently appears and disappears, mobile,



Fig. 24.29: Intussusception.

Volvulus

Volvulus is twist of loop of the bowel. It could be gastric, midgut, caecal or sigmoid. Sigmoid volvulus is *commonest* type. *Caecal volvulus* is more common in young people, common in females, rotates in clockwise direction. It is common in pregnancy. *Sigmoid volvulus* is common in elderly males, is anticlockwise rotation. It should rotate more than $1\frac{1}{2}$ turn to cause volvulus. *Predisposing factors* are adhesions, peridiverticulitis, loaded pelvic colon, long pelvic mesocolon, narrow attachment of sigmoid mesentery. Sudden pain, absolute constipation, enormous distension of abdomen, *tympanic abdomen*, dehydration, late faeculent vomiting, perforation and gangrene colon causing peritonitis are the features. Abdomen feels like a *tyre*. X-ray abdomen shows *omega sign* or *coffee bean sign* (Figs 24.30A and B).

Bolus Obstruction

Roundworms cause bolus obstruction in ileum. Pain, distension, vomiting are the features. Occasionally bolus mass may be felt in the right iliac fossa. Roundworm can perforate inflamed ulcerated bowel causing peritonitis.

Gallstone ileus is due to cholecystoduodenal fistula wherein gallstones roll down towards ileum causing bolus obstruction.



Figs 24.30A and B: Sigmoid volvulus – gangrenous.

Food bolus is formed by dry fruits, coconut, orange pulp, undigested vegetables which obstruct at 24 cm proximal to ileocaecal valve (narrowest part).

Meconium pellets, stercoliths, foreign body, tricho-bezoars are *other causes* of bolus obstruction.

Acute Salpingitis

It is the inflammation of Fallopian tubes. Pain begins at hypochondrium later spreads to iliac fossa. Scalding

pain in the urethra, fever, tenderness in iliac fossa, mass, vaginal discharge, tender cervix on per vaginal examination are the features.

Acute Nonspecific Mesenteric Lymphadenitis

It occurs in children below the age of 6 years occasionally can occur up to 14 years. Pain in right iliac fossa and umbilicus; fever, vomiting, tenderness in right iliac fossa are the features. Guarding is not obvious; tenderness elicited in supine position will shift towards left once patient is kept in left side position—*Klein's sign*. Rebound tenderness is absent. Occasionally on deep palpation tender lymph nodes may be felt.

Ruptured Ectopic Gestation

Sudden severe pain in the abdomen, distension, pallor, tachycardia, tachypnoea, missed menstrual period, discolouration around the umbilicus, tenderness in iliac fossa, shifting dullness, rebound tenderness, soft tender cervix, dropping haemoglobin, positive urine pregnancy test are the features. US confirms the diagnosis.

Ruptured Aortic Aneurysm

It may be intraperitoneal or extraperitoneal (retroperitoneal). Retroperitoneal rupture causes sudden severe pain in the abdomen, back pain, pallor, shock, tenderness, rigidity of abdomen. Blood clot may be felt like a mass in the iliac fossa. Condition often mimics acute pancreatitis, perforation of bowel, myocardial infarction.

Munchausen's Syndrome

(*BHK F von Munchausen 1797 German officer who used to tell tall stories of war between Russia and Turks*)

It is abdominal malingering wherein patient tells long history of his disease like a story. His abdomen shows several scars of earlier surgeries. It may be abdominal, bleeding or with faints, fits, palsies, etc. Appendicectomy is the common initial operation which patient undergoes.

Colicky pain

It is gripping pain for certain period, subsides for sometime and suddenly reappears again in similar fashion. Associated with nausea, vomiting, belching, retching, tachycardia, but abdomen is soft, guarding and tenderness is less compared to severity of pain. It can be:

Ureteric colic due to ureter stone.

Biliary colic (gallbladder colic due to spasm of gallbladder to force the stone).

Intestinal colic due to toxic enteritis, infection, lead colic in painters.

Appendicular colic in obstructive appendicitis.

Medical conditions causing abdominal crisis

Diabetic crisis presenting as acute abdominal pain occurs just prior to development of diabetic coma due to very severe hyperglycaemia. Patient will be ketotic.

Porphyria crisis presents as violent intestinal colic with constipation, precipitated by barbiturates and sulphonamides (idiosyncrasy); distended abdomen without rigidity is the feature. Urine kept over night will show dark red colour.

Hyperlipidaemia crisis causes left sided abdominal pain and is familial.

Other causes: Malaria, sickle cell crisis (splenic destruction—autosplenectomy; mesenteric vessel occlusion), haemophilia, acholuric jaundice, Tabetic crisis (gastric crisis).

Common causes of acute abdomen in children

Acute appendicitis

Intussusception

Intestinal obstruction due to roundworms, bands and adhesions

Acute non-specific mesenteric lymphadenitis – below 6 years

Meckel's diverticulitis

Primary acute peritonitis

Causes in females other than causes which are seen in both sexes

Ruptured ectopic gestation

Ruptured ovarian cyst

Torsion of ovarian cyst

Acute salpingitis

Tubo-ovarian abscess

Torsion or degeneration of uterine fibroid

Examination in Dysphagia

Dysphagia is a distressing symptom of many proximal gastrointestinal diseases. It is defined as difficulty in swallowing.

Aphagia is inability to swallow.

Odynophagia is painful swallowing.

Globus pharyngeus is sensation of a lump lodged in throat.

Phagophobia is fear of swallowing.

Sitophobia is fear of eating due to subsequent anticipated abdominal discomfort seen in mesenteric vascular insufficiency and regional ileitis.

Nausea is feeling of immense desire to eat.

Retching is laboured rhythmic contraction of respiratory and abdominal muscles that may proceed into vomiting eventually.

Hiccough (Hiccup/Singultus) is sudden spasmodic involuntary contraction of diaphragm with closed glottis causing short sharp inspiratory sounds.

Regurgitation is appearance of previously swallowed food in the mouth without vomiting.

Water brash is sudden filling of mouth with saliva as a reflux response.

Heart burn/pyrosis is sensation of burning or warmth substernally or in the high epigastrium which radiates into the neck and arms.

Belching is chronic repetitive eructations.

cardia, oesophageal webs are common in middle aged. Carcinoma of oesophagus is common in old age. Hysteria causing dysphagia is common in young girls.

Sex

Sideropenic dysphagia due to oesophageal webs (Plummer-Vinson syndrome) is seen in females. Carcinoma oesophagus is common in males.

Mode of Onset

Sudden onset of dysphagia is seen in foreign body impaction (fish bone, food bolus, solid materials) in children; acute inflammation of pharynx, retropharyngeal area, oesophagus. *Slowly progressive for long period* is a feature of benign disease like achalasia, benign stricture. *Rapidly progressive dysphagia of short duration* is a feature of carcinoma of oesophagus. In achalasia, to begin with dysphagia is mainly to liquids as weight of solid food opens up the area of spasm to relieve dysphagia. In carcinoma oesophagus first it is for solid, later to liquid. Food getting stuck is classical history but site of blockage is difficult to assess even though patient points out the site.

Regurgitation is common in achalasia, pharyngeal pouch, sliding hiatus hernia.

Pain

It is mainly discomfort but often pain may be predominant symptom. It is commonly observed in reflux oesophagitis, corrosives. Pain may be under the sternum.

Vomiting

Nature of the vomitus, haematemesis, content, smell, should be asked for. Paraoesophageal hernia causes post-prandial vomiting.

HISTORY

Present History

Age

Oesophageal atresia, dysphagia lusoria are seen in infants. Foreign body, diphtheria, retropharyngeal abscess are common in children. Stricture, achalasia

Cough

When there is obstruction in the oesophagus, food, liquid, saliva may aspirate into the lungs causing aspiration, bronchopneumonia. Achalasia, pharyngeal pouch, carcinoma may cause cough. Tracheo-oesophageal fistula causes severe cough after feeding.

History of Dyspnoea, Voice Change

Mediastinal compression by mediastinal lymph nodes or other masses can cause dyspnoea or stridor. Lymph node compressing the recurrent laryngeal nerve also can cause hoarseness of voice. Hoarseness of voice may be due to advanced carcinoma of pharynx where dysphagia is also main symptom.

Loss of Appetite and Loss of Weight

It is a feature of carcinoma of oesophagus. Weight loss is also a feature of achalasia cardia.

Past History

Past history of surgery, chemotherapy, radiotherapy for carcinoma, neck pathology should be asked. Past history of vagotomy may be the cause for dysphagia. Treatment for hiatus hernia earlier is important.

Personal History

Smoking, alcohol and diet history is important in carcinoma oesophagus.

General Examination

Patient will be emaciated with significant weight loss in carcinoma oesophagus. Glossitis, atrophied smooth, pale tongue is common. Pulse on both wrists will be different and not equal in aneurysm of aorta. Clubbing may be evident if there is associated lung pathology. Tonsils may be site of the primary causing dysphagia. Pharynx should be examined for retropharyngeal abscess, ulcers.

Examination of Neck

It is very important to examine the neck. Neck is examined for thyroid, lymph node mass and pharyngeal pouch. Pharyngeal pouch is a soft swelling on the left side of the neck; on pressing the swelling patient

develops regurgitation. Left supraclavicular area should be examined for lymph node enlargement.

Standing behind the patient examiner holds the cricoid cartilage with an upward traction. Downwards tug can be felt with each aortic pulsation – ‘*tracheal tug*’ is a feature of aneurysm of arch of aorta.

Examination of the Chest

Respiratory system is examined for pleural effusion, pneumonia, lung abscess, tracheal displacement, dullness over the sternum on percussion.

Abdominal Examination

Liver may be enlarged and is nodular, hard due to secondaries from lower third oesophageal carcinoma. Rarely OG junction mass may be palpable in the epigastrium.

Examination of Spine

Tuberculosis of spine can cause dysphagia by abscess at retropharyngeal region or mediastinum.

Dysphagia

Dysphagia is difficulty in swallowing. Painful swallowing is *odynophagia*. It can be *acute*—due to foreign body or acute infection or *chronic* due to causes like stricture or carcinoma, etc. Associated hoarseness of voice may be present in advanced pharyngeal or post cricoid carcinomas. Laryngeal carcinoma at late stage also can cause dysphagia along with hoarseness of voice. Dysphagia can be *oropharyngeal* or *oesophageal* depending on the cause. Dysphagia may be due to pathology in *voluntary/pharyngeal phase* of the swallowing wherein patient also develops coughing while swallowing. Dysphagia due to problem in *oesophageal involuntary phase* of swallowing is specified by food getting stuck in the pathway. But site of “*food getting stuck*” feeling is not relevant. Dysphagia can be *progressive or intermittent*.

Causes of Dysphagia

Common Causes

Gastro-oesophageal reflux diseases (GORD/GERD); Hiatus hernia;

Causes of dysphagia		
Extraluminal causes	Causes in the wall of oesophagus or other area	Causes in the lumen
Mediastinal nodes – secondaries / lymphoma / tuberculosis Aortic aneurysm Rolling hiatus hernia Thyroid enlargement – malignant Dysphagia lusoria Congenital anomalies Mediastinitis / mass	Carcinoma oesophagus Corrosive / tuberculous / inflammatory / congenital stricture oesophagus GERD Achalasia cardia Oesophageal candidiasis Plummer-Vinson syndrome Oesophageal diverticulum Carcinoma posterior 1/3 of tongue / pharynx Diffuse oesophageal spasm Congenital anomalies Retropharyngeal abscess / peritonsillar abscess (Quinsy) / acute tonsillitis / pharyngitis	Foreign body in the oesophagus – coin/ dentures / fish or meat bone Other causes Cranial causes (neurological) – Bulbar palsy / infarction / hemiplegia Vertebrobasilar insufficiency

Carcinoma oesophagus—Here dysphagia is of short duration and progressive. 2/3 of the lumen should be blocked by tumour to develop dysphagia;

Foreign body oesophagus—It may be coin/bone piece/denture. It is common in children. It causes acute dysphagia. It may be life threatening often;

Carcinoma of pharynx or posterior 1/3 of the tongue;

Corrosive strictures—It is usually alkali stricture. Squamous mucosa has resistance to acid effect to certain extent;

Oesophageal candidial infection—It is becoming common due to immunosuppression in association with HIV infection; steroid therapy; cancer chemotherapy; post-transplant period, etc. Presentation is dysphagia and odynophagia. Oral candidiasis (thrush) is obvious. Endoscopy shows whitish curd-like plaques in the oesophageal mucosa which can not be moved (food particles can be moved) (**Fig. 25.1**). Barium swallow shows mucosal ulceration and irregular areas. Biopsy confirms the diagnosis. Treatment is oral antifungal as well as topical antifungal therapy.

Plummer-Vinson syndrome

Mediastinal swellings—Primary tumours/nodal mass either lymphoma or secondaries or tuberculosis.

Rare Causes

Diffuse oesophageal spasm: They are in coordinated contractions of oesophagus causing chest pain or dysphagia. It is common in distal 2/3 of the oesophagus.



Fig. 25.1: Endoscopic view of the oesophageal candidiasis infection. Note the whitish curd like plaques.

Hypertrophy of circular muscle fibers with very high persistent pressure of 400-500 mm Hg is specific. Treatment is calcium channel blockers, vasodilators, endoscopic dilatation and extended oesophageal surgical myotomy up to the aortic arch (very useful especially for dysphagia; not much for chest pain). Oesophageal diverticula, Chaga's disease.

Dysphagia lusoria: It is a congenital vascular anomaly of aortic root. Aortic arch anomalies are – double arch (40%), right arch and left ligamentum arteriosum (25%), anomalous innominate or common carotid artery or aberrant right subclavian artery (10%). It is due to disappearance of proximal right 4th aortic

arch instead of distal portion. All patients having this anomaly (*dysphagia lusoria*) have got an *aberrant right subclavian artery* in a transposed position arising from descending aorta that courses posterior to oesophagus. Often there will be a complete *vascular ring* around trachea and oesophagus. It is categorised based on their specific subclavian anomaly – depends on presence of aneurysm, occlusive disease and compression. Commonly they are asymptomatic. Presentations may be dysphagia, chest pain, stridor, wheeze, recurrent respiratory infection (usually presents after the age of 40). Investigations: CT chest, MRI, chest X-ray, barium swallow (T4 level diagonal impression) and endoscopy—(Shows pulsating extraluminal compressive mass).

Thyroid swelling: It is uncommon to develop dysphagia in a thyroid swelling. There will be always dyspnoea when dysphagia develops. Large malignant thyroid or anaplastic thyroid can cause dysphagia with dyspnoea or stridor.

Boerhaave's syndrome: It is vertical full thickness tear of lower oesophagus due to vomiting with closed glottis. It is often life threatening and emergency.

Neurological causes like stroke, bulbar palsy, motor neuron disease, Parkinson's disease, etc.

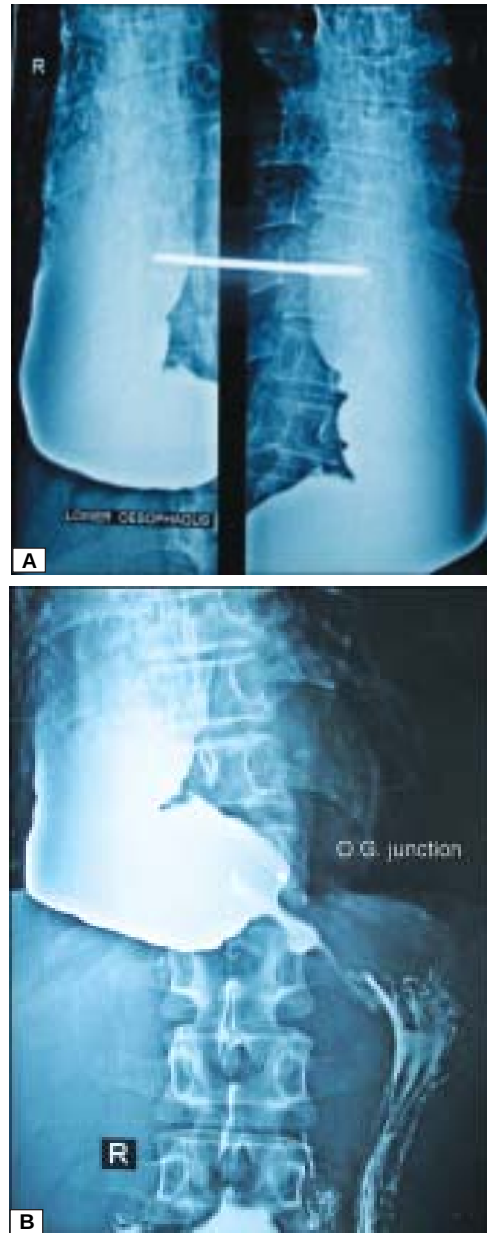
Congenital anomalies of oesophagus; Mediastinal fibrosis.

Drug induced dysphagia: Drugs like KCl, quinine, NSAID can cause dysphagia.

Evaluation of a Patient with Dysphagia

Proper history; Haematocrit; Chest X-ray often may show mediastinal mass lesion/foreign body. *Oesophagoscopy:* Once lesion is detected, it is treated accordingly. Biopsy from lesions, endotherapy if needed should be carried out (like F/B removal; stricture dilatation; sclerotherapy). *Rigid Negus* oesophagoscope is used to remove foreign body under general anaesthesia. *Fiberoptic/video flexible* oesophagoscope is used for diagnosing and taking biopsy.

Barium swallow may show irregular filling defect or extrinsic compression. Water soluble contrast is used in suspected perforation or fistula. Achalasia cardia shows 'bird beak' oesophagus with proximal dilatation (**Figs 25.2A and B**). *Corkscrew* oesophagus



Figs 25.2A and B: Barium swallow showing dilated oesophagus (sigmoid oesophagus/megaoesophagus) in achalasia cardia.

is a feature of diffuse oesophageal spasm. Shouldering and irregular filling defect is a feature of carcinoma of oesophagus.

CT scan chest is very useful method to identify the anatomical location of the cause (nodes/tumour/

aorta/cardiac cause/congenital). Extent, spread, nodal status, size and operability of tumour also well assessed. *Oesophageal manometry* is done in Achalasia cardia/GERD. *24 hours pH monitoring* is ideal and most accurate for GERD. Small pH probe (transnasal catheter) is passed in the distal oesophagus 5 cm proximal to upper margin of LOS under manometry guidance. Probe is connected to a digital recorder worn by the patient for 24 hours. Record is analyzed using a computer. A pH less than 4 for more than 4% of total 24 hours period (more than near to one hour in toto in 24 hours) is pathological reflux. It is often assessed by scoring system. Radio telemetry pH probes are used now without any nasal tube. It is passed and placed on the oesophageal wall using endoscope.

Endosonography is very useful in many conditions causing dysphagia. It can assess site, layers of the oesophagus, nodes, spread, etc. properly. Different layers are seen as alternating hyperechoic and hypoechoic bands.

Ultrasound abdomen to see abdominal nodes/liver/ascites; MRI study.

Endoscopic oesophageal staining using labeled iodine is used to identify early carcinoma in oesophagus. Normal mucosal cells contain glycogen which takes iodine and so stains brown, whereas carcinoma cells will not take up iodine and so mucosa appears pale (not stained) (**Fig. 25.3**).



Fig. 25.3: Endoscopic view of carcinoma of oesophagus.

OEESOPHAGEAL MOTILITY DISORDERS

Neurological – stroke, bulbar palsy, motor neuron disease, multiple sclerosis, Parkinson's disease
Muscular – myasthenia, muscular dystrophy
Pharyngeal pouch
Diffuse oesophageal spasm
Nutcracker oesophagus
Autoimmune disorders like systemic sclerosis, polymyositis and CREST syndrome
Reflux associated
Idiopathic
Eosinophilic allergic oesophagitis
Achalasia cardia
GERD
Alcoholic neuropathy

Foreign Body in the Oesophagus

Common foreign bodies are—Coins; Dentures; Pins; Fish or meat bones (**Fig. 25.4**).



Fig. 25.4: X-ray showing foreign body in oesophagus—coin.

Sites of impaction in oesophagus: Cervical constriction—C₆; Bronchoaortic constriction—T₄; Diaphragmatic constriction—T₁₀; Pre-existing malignancy or inflammatory stricture site. Features—Sudden dysphagia with chest pain and breathlessness. Later features of

shock, sepsis, mediastinitis, empyema. *Rigid oesophagus* is used to extract foreign body.

Gastro-oesophageal Reflux Disease (GORD/GERD)

It is a pathological reflux from the stomach into the lower oesophagus.

Anatomical Factors

Obesity, altered length of intra-abdominal oesophagus, altered obliquity of O-G junction, reduced pinching action of crus of diaphragm.

Physiological Factors

Reduced Lower Oesophageal Sphincter pressure, altered transient relaxation period in L O S, reduced oesophageal clearance mechanism. Delayed gastric emptying due to diabetes, neuromuscular block, gastroparesis, medications; increased gastric distension and gastric acid hypersecretion are other features.

Other Factors

Alcohol, smoking, stress, lifestyle.

Types

Symptomatic uncomplicated GORD and symptomatic, complicated GORD.

Clinical Features

Fatty dyspepsia; Odynophagia; Appearance of symptoms within seconds of ingestion of food is typical; Chest pain and heart burn (pyrosis); epigastric pain, regurgitation; laryngeal symptoms; dysphagia will occur once complications begin; symptoms are more with change of position; Chronic cough, shortness of breath and hoarseness.

Complications

Reflux oesophagitis; sliding hiatus hernia; stricture lower end oesophagus; oesophageal shortening; Barrett's oesophagus; carcinoma (adeno) oesophagus.

Differential Diagnosis

Achalasia cardia; carcinoma oesophagus; peptic ulcer; gallstones; pancreatic diseases; gastritis.

Barrett's Oesophagus

It is metaplastic changes in the mucosa of the oesophagus in response of GORD. Squamous epithelium of lower end of the oesophagus is replaced by diseased columnar epithelium (columnar metaplasia). There is macroscopic visible length of columnar mucosa with microscopic features of intestinal metaplasia. If the length of metaplasia is more than 3 cm. It is called as *long segment Barrett's oesophagus*. If the length is less than 3 cm it is called as *short segment Barrett's oesophagus*. This diseased columnar epithelium is more prone for malignant transformation, i.e. when there is intestinal metaplasia, risk of malignant transformation increases that too when there is more amount of dysplasia.

Barrett's Ulcer

It is an ulcer in columnar lined Barrett's oesophagus at or just above the squamo-columnar junction. It is more prone for *bleeding, perforation, adenocarcinoma of oesophagus*.

Hiatus Hernia

It may be sliding hernia (85%), Rolling hernia (10-12%), Combined (**Fig. 25.5**).

Sliding hernia is commonly associated with GORD (Should be discussed like GORD).

Types of hiatus hernia – classification

Type I hiatus hernia—It is the cephalad displacement of the gastrooesophageal junction through the hiatus into the mediastinum. It is usually small, asymptomatic and reducible. It is commonest type

Type II hiatus hernia—It is superior migration of the fundus of the stomach along side the GE junction and oesophagus into the mediastinum with GE junction in normal intra-abdominal location. It is rolling hernia

Type III hiatus hernia—It is combination of type I and type II

Type IV hiatus hernia—It is the hernia containing other abdominal viscera as content like transverse colon and omentum

Rolling Hernia (Paraesophageal Hernia)

It is herniation of stomach or rarely other abdominal contents through a hiatus usually towards left side.

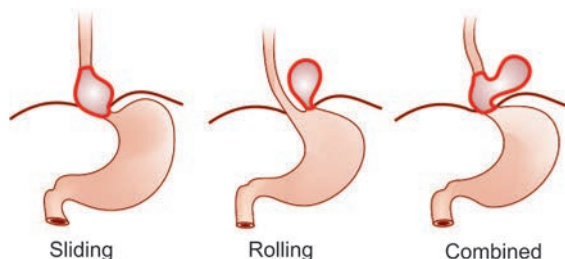


Fig. 25.5: Types of hiatus hernia.

Clinical features:	Complications:
Pain	Gangrene of stomach.
Hiccough	Perforation into the mediastinum.
Regurgitation	Perforation into the peritoneum.
Cardiac abnormality.	Gastric volvulus.

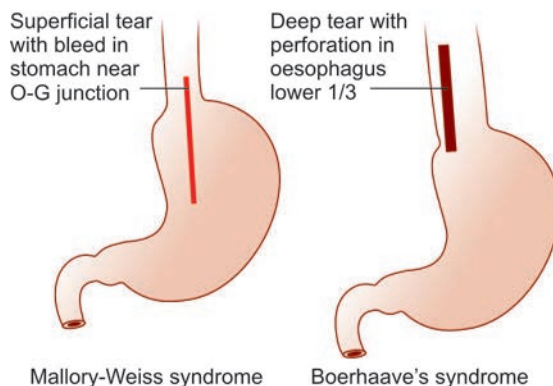


Fig. 25.6: Diagram showing Mallory-Weiss syndrome and Boerhaave's syndrome.

Boerhaave's Syndrome

It is a tear in the lower third of the oesophagus which occur when a person vomits *against a closed glottis*, causing leak into the mediastinum, pleural cavity and peritoneum. Crunching effect in the chest is called as *Hammam's sign*. *Meckler's triad*: Vomiting; chest pain; subcutaneous emphysema. Chest X-ray shows pneumomediastinum—'V' sign of Nacleario.

Mallory-Weiss Syndrome

It is seen in adults with a severe prolonged vomiting causing longitudinal tear in the mucosa of stomach at and just below the cardia, *leading to severe haematemesis*. Violent vomiting often may be due to migraine or vertigo. It is superficial mucosal tear mainly of proximal stomach and often of lower oesophagus (10%) at one O'clock position. Often presents with severe vomiting and later haematemesis, with features of shock (Fig. 25.6).

Tracheo-oesophageal Fistula (TOF)

Types

In 85% cases it is a blind upper end with lower end communicating with trachea. It may be associated with VACTER anomalies.

Clinical Features

TOF should be recognized within 24 hours of birth. Newborn baby regurgitates all feeds and there is continuous pouring of saliva from the mouth which is a *diagnostic feature*. It is commonly associated with maternal *hydramnios* (50%). Reveals obstruction while passing nasogastric tube. Contrast study will reveal fistula and obstruction. Associations—VACTER anomalies (Fig. 25.7).

VACTER anomalies

V—Vertebral defects.
A—Anal atresia.
C—Cardiac defect (PDA/VSD).
TE—Tracheo-oesophageal fistula.
R—Radial hypoplasia and renal agenesis.

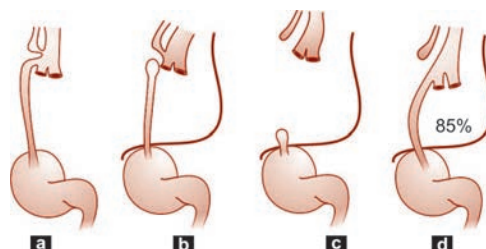


Fig. 25.7: Types of tracheo-oesophageal fistula: (a) H type, (b) Lower end blind, upper end connected to trachea, (c) Both ends blind, (d) Upper end blind, lower end connected to trachea (85%).

Presentations	Differential diagnosis	Complications
Sudden onset of symptoms Severe chest pain Pain abdomen Shock	Myocardial infarction Pancreatitis	Mediastinitis Septicaemia.

Achalasia Cardia

It is failure of relaxation of cardia (Oesophago gastric junction) due to disorganised oesophageal peristalsis, due to failure of integration of parasympathetic impulses causing *functional obstruction*.

Aetiology

Stress, Vit B₁ deficiency, Chaga's disease. There is pencil-shaped narrowing of cardia (O-G junction) with enormous dilatation of proximal oesophagus, which contains foul smelling fluid and is more prone for aspiration pneumonia. Achalasia cardia is a *precancerous* condition.

Clinical Features

Common in females between 20-40 years age group, present with progressive dysphagia, which is more for liquid than to solid food. Regurgitation and recurrent pneumonia are common. *Triad*: Dysphagia; regurgitation; weight loss. Malnutrition and general ill health is common.

Investigations

Barium swallow is diagnostic—shows (1) Pencil-like smooth narrowing of lower oesophagus. (2) Dilatation of proximal oesophagus (sigmoid oesophagus). (3) Absence of fundic gas bubble. Chest X-ray shows patches of pneumonia. Oesophageal manometry shows



Fig. 25.9: Barium swallow X-ray showing achalasia cardia.

unrelaxed lower sphincter of oesophageal sphincter. Oesophagoscopy is done to confirm the diagnosis and to rule out carcinoma oesophagus (**Figs 25.8 and 25.9**).

Corrosive Stricture of Oesophagus

Mainly due to alkali - sodium hydroxide, occasionally due to acid (sulphuric acid, nitric acid, etc). Acid commonly damages the stomach. It causes extensive inflammation of the mucosa with periesophagitis which if not treated will lead to multiple strictures in oesophagus. Sometimes it causes severe life threatening necrotising lesion which requires immediate surgical intervention.

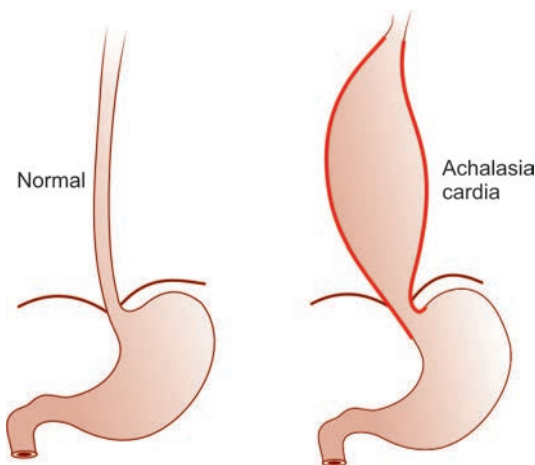


Fig. 25.8: Achalasia cardia—diagrammatic look.

Causes of stricture oesophagus

Peptic stricture (oesophagitis induced).
Corrosives—commonest cause.
Foreign body
Post-surgical
Congenital
Infection like tuberculosis.
Drugs like tetracycline, vitamin C

Plummer-Vinson Syndrome (Paterson-Kelly Syndrome)

(1) *Oesophageal webs* seen in uppermost portion of oesophagus with spasm of circular muscle fibers. It is a premalignant condition. (2) *Iron deficiency anaemia*. (3) *Superficial glossitis*, cheilitis, *kolionychia*. (4) Splenomegaly. In oesophageal webs mucosa is hyperkeratotic, friable, desquamated and causes severe dysphagia. Oesophagoscopy and biopsy is required to rule out malignancy.

Reflux Oesophagitis

Types

1. *Acute*: Following burns, trauma, infection, peptic ulcer.
2. *Chronic*: Reflux of acid in sliding hernia, after gastric surgery.

Reflux is quite common in pregnancy. Site is always in lower oesophagus.

Pathology

There is bleeding granulation tissue in lower oesophageal mucosa with spasm of longitudinal muscle which pulls the adjacent gastric area upwards into the oesophagus causing sliding hernia.

Clinical Features

It is a part of GORD; Pain and burning sensation in retrosternal area often referred to shoulder, neck, arm, etc. Heart burn is common; dysphagia; anaemia.

Grading:

- 1) Mucosal erythema.
- 2) Mucosal erythema + superficial ulceration.
- 3) Mucosal erythema + superficial ulceration + submucosal fibrosis.
- 4) Mucosal erythema + extensive ulceration + paramural fibrosis.

Schatzki Rings

They are semicircular protrusion of lower oesophageal mucosa located at or just above the oesophago-gastric junction (squamo-columnar junction). Its undersurface is lined by columnar gastric epithelium. They involve only the mucosa and submucosa not the muscle. They present with dysphagia and reflux.

Carcinoma Oesophagus

Aetiology

Diet, deficiencies	5% common
Mycotoxin	Common after 45 years
Alcohol and tobacco	Common in men
Achalasia cardia	Common in China-Henan
Oesophageal webs	province

Tylosis

Autosomal dominant condition seen from childhood. Soles and palms are involved called as *palmo-plantar keratoderma*.

Waxy, yellow lesions, which does not itch.

60% of members of families develop carcinoma oesophagus after the age of 60.

Systemic retinoids are the drugs used for tylosis.

Pathology

Common in middle third—50%—Squamous cell carcinoma. Lower third—33%—Adenocarcinoma. Upper third—7%—Squamous cell carcinoma. Lower 3 cm of oesophagus is lined by columnar epithelium, and so adenocarcinoma is common here. Barrett's columnar metaplasia, which occurs in lower third oesophagus is also more prone for adenocarcinoma.

Gross

Annular; Ulcerative; Fungating—cauliflower-like.

Spread

Direct: In upper third it spreads through muscular layer and get adherent to left main bronchus, trachea, and left recurrent laryngeal nerve (causes hoarseness), aorta or its branches (causes fatal haemorrhage, but rare). It may perforate and cause mediastinitis. It may get adherent to pleura also. *Lymphatic spread*: It spreads both by lymphatic permeation and lymphatic embolisation. It can cause satellite nodules elsewhere in the oesophagus away from the main tumour. Above in the neck it spreads to left supraclavicular lymph nodes. In the thorax it spreads to para-oesophageal, tracheobronchial lymph nodes to subdiaphragmatic lymph nodes. In the abdomen it spreads to coeliac lymph nodes. *Blood spread occurs* to liver.

Clinical Features

Recent onset of dysphagia is the commonest feature. For the dysphagia to develop two-third of the lumen should be occluded. Regurgitation; anorexia and loss of weight (severe, cachexia); Pain—substernal or in the abdomen; Liver secondaries; Bronchopneumonia; Features of broncho-oesophageal fistula in carcinoma of upper third oesophagus; Supraclavicular lymph nodes may be palpable.

Investigations

Barium swallow: Shouldering sign and irregular filling defect;. Oesophagoscopy to see the lesion, extent and type (**Fig. 25.10**);. Biopsy for histological type and confirmation;. Chest X-ray to see pulmonary infection;. Bronchoscopy, to see invasion in upper third growth;. *Oesophageal endosonography* to look for the involvement of layers of oesophagus; CT scan to look for local extension and status of tracheobronchial tree in case of upper third growth; US abdomen to look for liver and lymph node status in abdomen; Endoscopic oesophageal staining with labeled iodine will result in normal mucosa being stained brown, but remains pale in carcinoma (as ca mucosa will not take up iodine).

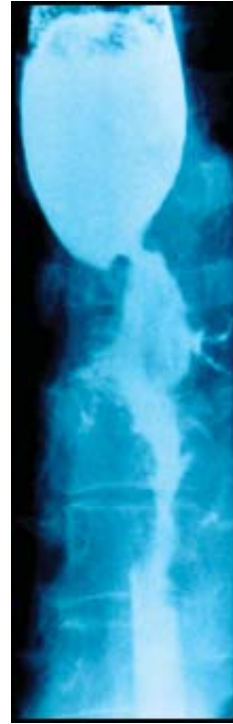


Fig. 25.10: Barium swallow X-ray showing irregular filling defect and shouldering sign in midoesophagus—carcinoma oesophagus.

Examination of Chest Diseases

This chapter is important for systemic management of surgical problems. Preoperative respiratory assessment and postoperative respiratory care is very important. Complications like pneumonia, collapse of lung, empyema, aspiration, ARDS (Acute respiratory distress syndrome), pulmonary embolism, pneumothorax, haemothorax, pleural effusion, lung abscess, postoperative cardiac problems like myocardial infarction should be remembered.

Per se patient may be having bronchogenic carcinoma, mediastinal pathology, pulmonary tuberculosis, asthma, bronchitis, bronchiectasis and so detailed history taking, clinical examination, relevant investigations should be done.

History

Chest pain: It is the commonest symptoms in most of the lung and cardiac diseases. It may be heaviness in chest in early carcinoma lung or pain in shoulder, intercostal region (intercostal neuralgia) in advanced disease.

Fever, dyspnoea, haemoptysis, cough (dry or with sputum), nature of sputum, productive or not, foul smelling or not. Tuberculosis is the commonest cause of haemoptysis in India.

Loss of weight and appetite are the features of tuberculosis and carcinoma of lung.

Swelling chest wall: It could be cold abscess, empyema necessitans, and tumour.

Discharging sinus formation could be tuberculosis or actinomycosis.

Previous history of tuberculosis, pneumonia, hospitalisation, surgery are important.

Smoking history is very relevant in lung diseases.

General Examination

Assessment of anaemia, clubbing, dyspnoea, nutritional status is important.

Local Examination

Inspection

Shape of the Chest Wall

(1) *Scoliosis*, thoracic kyphoscoliosis reduces the pulmonary ventilatory capacity. (2) *Funnel chest* also called as *pectus excavatum* is a condition wherein there is posterior concavity of sternum from above downwards, anteroposteriorly and side to side of the chest. (3) *Flat chest* where transverse diameter is more than anteroposterior diameter. (4) *Barrel chest* where anteroposterior diameter is more than transverse diameter. (5) *Ricketic chest* with bead-like prominences at costochondral junction – *Ricketic rosary*. (6) *Pigeon chest/Pectus carinatum/chicken breast* where sternum is prominently bowing forward with costal cartilages; ribs indrawn along with symmetrical Harrison's sulci/grooves. It is seen as congenital deformity or as a sequel of childhood respiratory diseases. (7) Swelling or sinus in the chest wall should be examined in detail (**Figs 26.1A and B**).

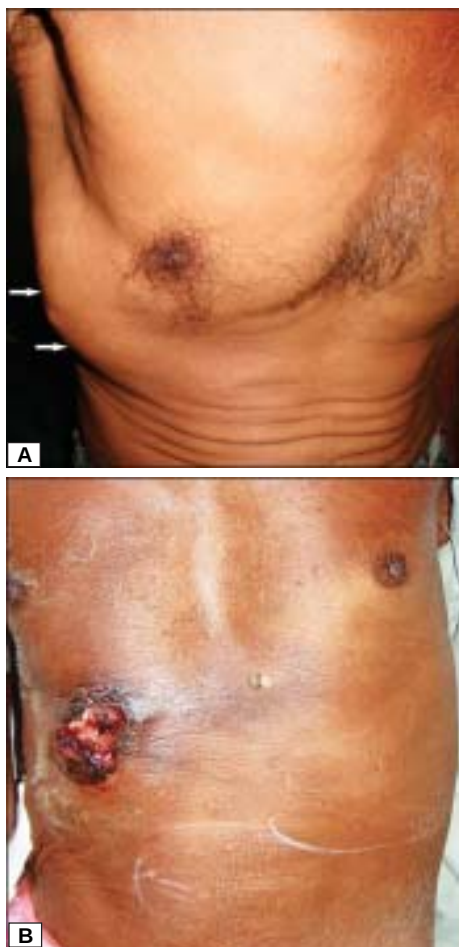
Respiratory movements: Rate, equality, character, type should be observed.

Apex beat should be checked.

Neck veins may be engorged in congestive cardiac failure, pulmonary hypertension, and pericardial effusion. Neck also should be inspected for enlarged lymph nodes (swelling).

Palpation

Respiratory movements are checked by placing fingers of each hand on each side of the chest wall of the



Figs 26.1A and B: Chest wall should be examined for swelling, ulcer.

patient with thumbs placed at the center and tips of thumbs touching each other. Patient is asked to take deep breath. Defective movements are appreciated.

Position of the trachea whether shifted or not is noted.

Vocal fremitus is noted by placing flat of fingers over chest wall. It is reduced in effusion/pneumothorax; increased in consolidation.

Swelling over chest wall should be palpated in detail. Impulse on coughing is due to empyema necessitans. *Ribs and relation of swelling* to them should be assessed. Expansile pulsation may be due to an aneurysm. Sinus also should be examined for all features in detail.

Percussion, auscultation are very essential methods to elicit all findings in respiratory (effusion, consolidation, pneumothorax, emphysema, etc.) and cardiac systems.

Palpation of neck nodes and spine is done for any significant pathology.

Investigations

Chest X-ray, bronchography (using neohydriol contrast into the bronchial tree), barium swallow (to see compression) (**Fig. 26.2**).

Bronchoscopy to look for tumour in the bronchus (not in the periphery of lung), to take brush biopsy/direct biopsy, to do bronchial wash, look for vocal cord palsy by tumour infiltration of nodes.

Mediastinoscopy by making a small incision above the sternum.

Laryngoscopy to see vocal cord palsy.

CT chest is essential investigation. CT guided biopsy can be done.

Pleural tap is simple easier good investigation.

Scalene node biopsy.

Lung function tests.

Thoracoscopy is very useful method.

Metastatic work up for carcinoma lung towards brain, liver, bones.



Fig. 26.2: Massive malignant pleural effusion right sided – in a chest X-ray.

Empyema Thoracis

It is collection of pus in pleural cavity.

Causes

An empyema is never primary. Secondary causes are:—

(a) From chest wall: Wounds, osteomyelitis of ribs.
 (b) From lung: pneumonia, abscess, bronchiectasis, tuberculosis, growth.
 (c) Postoperative: After thoracotomy.
 (d) From oesophagus: Perforations, carcinoma.
 (e) From below diaphragm: Subphrenic abscess.
 Tuberculosis and pneumonia are common causes in developing countries.

Pathology

Initially serous fluid collects which eventually becomes purulent. Intrapleural clotting of pus occurs with thickening of pleura and later fibrinous adhesion forms resulting in matured *empyema*. Chest is drawn inwards and is immobile; mediastinum is drawn inwards and diaphragm gets elevated. It leads into rigid contracted immobile chest with functionless lung underneath - *Frozen chest*. Often pus will perforate through intercostal space and forms *empyema necessitans*.

Organisms

Initially staphylococci, streptococci, pneumococci, and later *E.coli*, pseudomonas, drug—resistant staphylococci.

Clinical Features

Pain in the chest, tenderness, fever along with difficulty in breathing. Toxicity is seen in acute type of empyema. Dullness on percussion, absence of breath sounds decreased chest wall movements are the chest signs.

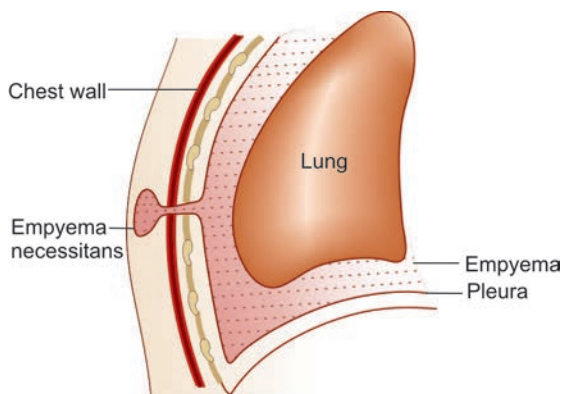


Fig. 26.3: Empyema necessitans.

Chest X-ray, CT scan, aspiration are the needed investigations to be done (Fig. 26.3).

Features of Empyema necessitans

Bulge in the intercostal space.
 Restricted movement.
 Tenderness.
 Dullness without breath sounds.
 Impulse on coughing

Lung Abscess

It is localised suppuration in the lung with tissue necrosis. It is end stage of suppurative pneumonitis with thrombosis of associated artery.

Aetiology: (a) *Infection:* Pneumonias due to streptococcus, staphylococcus, pneumococcus, haemophilus, anaerobic and other bacteria. (b) *Bronchial obstruction* due to tumours or foreign body. (c) *Chronic upper respiratory infection* due to sinusitis, tonsillitis and dental infection (anaerobic infection). (d) *Septicaemia*. As pus accumulates tension increases inside the abscess

Clinical types:

Acute empyema.
 Acute fulminant toxic empyema.
 Subacute empyema.
 Chronic empyema.
 Latent empyema.
 Persistent empyema.
 Empyema necessitans.
 Chronic empyema with sinus.
 Interlobar empyema.

Pathological types:

Exudative.
 Fibrinopurulent.
 Organising.

Anatomical types (Depending on the anatomical location):

Apical.
 Mediastinal.
 Interlobar.
 Diaphragmatic.
 Lateral

cavity causing spread into other areas of the lung or may rupture into the bronchus.

Clinical features: Acute onset of fever, but often it is recurrent in nature. Cough with expectoration is present. Haemoptysis with foul smelling sputum is often seen. Also associated with chronic illness with debilitation.

Complications: (a) Spread into other areas of the lung; (b) *Metastatic cerebral abscess*—Lung abscess is the *commonest* focus for metastatic cerebral abscess, occurs as a result of pyaemic emboli through paravertebral veins. (c) Hemorrhage may be torrential is due to erosion of the vessel in the abscess wall. (d) Empyema thoracis (**Fig. 26.4**).

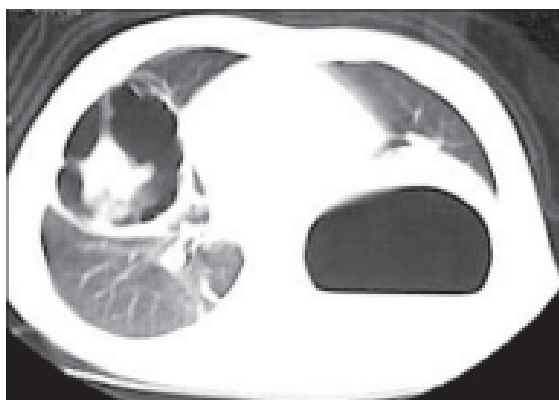


Fig. 26.4: CT scan showing lung abscess.

Mediastinal Tumours

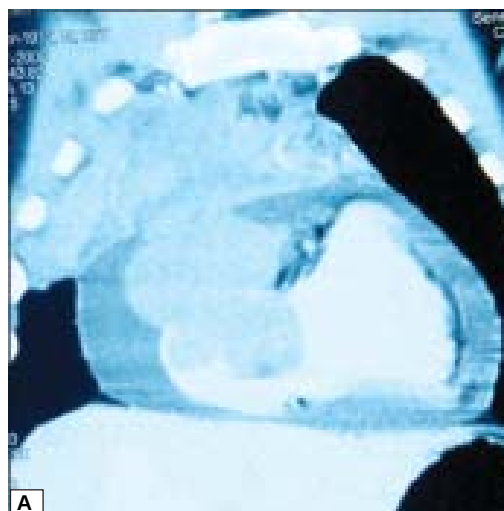
It occurs at any age group in both sexes. It is often picked up on routine chest X-ray, as about 50% are asymptomatic.

Presentations

Chest pain and back pain; respiratory distress; venous congestion; hoarseness of voice (due to compression over recurrent laryngeal nerve); dysphagia due to oesophageal compression; Horner's syndrome due to compression over sympathetic chain; Scabbard trachea; Later diaphragmatic paralysis may occur; Pleural effusion; haemorrhage due to erosion of major vessel by malignant tumour. Thymoma may present with myasthenia gravis (**Figs 26.5 and 26.6A and B**).



Fig. 26.5: X-ray showing mediastinal tumour – could be lymphoma.



Figs 26.6A and B: CT pictures showing mediastinal tumour.

Classification

Superior mediastinal tumour: Retrosternal goitre.

Anterior mediastinal tumours: Retrosternal goitre; Thymic tumours—thymomas are commonly associated with myasthenia gravis; Teratomas and dermoids; Pleuro-pericardial cyst.

Midmediastinal tumours: Lymphadenopathies of all causes; Foregut duplication cysts; Lipomas.

Posterior mediastinal tumours: Neurofibromas; Ganglioneuromas.

Shock Lung (Stiff Lung)

Causes

Major chest trauma; Septicaemia; Massive blood transfusions; DIC.

Pathogenesis

Development of microthromboembolism of small lung vessels following extensive intravascular coagulation leading into pulmonary consolidation which reduces the lung compliance markedly causing severe depression of gas exchange in the lung—a **stiff lung**. *It has got high mortality as lung cannot expand at all. Outcome is fatal if emergency treatment is not done.*

Pulmonary Embolism (PE)

It is due to deep venous thrombosis (DVT) which gets detached into circulation to cause pulmonary embolism. It may be of femoropopliteal or iliofemoral origin.

Types

Small emboli: Causes pulmonary hypertension.

Medium sized emboli lodge in branches of pulmonary artery causing chest pain, haemoptysis, dyspnoea.

Large (massive) emboli causes block in main pulmonary artery leading to sudden chest pain, severe dyspnoea, shock, raised venous pressure and **sudden death**. *Effects of Pulmonary Embolism:* Decreased cardiac output; Pulmonary vasospasm and pulmonary hypertension; Bronchospasm; Defective oxygenation of blood. *Risk Factors:* Postoperative and trauma patients who are bed ridden; pregnant women; Old age; Obesity and heart disease; varicose veins; all aetiologies which cause DVT; Carcinoma.

Clinical Features

Dyspnoea, chest pain and haemoptysis; tachycardia, tachypnoea and cyanosis; pleural rub and cardiac gallop.

Investigations

Chest X-ray PA view- Hyperlucency in an area of oligaemia - *Westermark sign*. CT scan and MRI – can detect PE. Pulmonary angiography is diagnostic (100%). Arterial blood gas analysis; *Isotope Radio-nuclide Ventilation – Perfusion (V/Q) Lung scanning:* While normal scan rules out PE, Evidence of V/Q mismatch is highly suggestive of pulmonary embolism. Doppler study and venography – To rule out DVT.

Carcinoma Lung

It originates from primary, secondary bronchus or from peripheral lung tissue. Smoking is the most common aetiology. Industrial toxins, old lung scar are other causative factors. Squamous cell carcinoma, small cell carcinomas are usual types. Chest pain, haemoptysis, hoarseness of voice due to recurrent laryngeal nerve palsy, weight loss, cachexia, metastases to lymph nodes in mediastinum, axilla and neck; spread to bones, brain and liver are the usual features. Pleural effusion and consolidation or collapse of lung can occur. Involvement of brachial plexus, intercostal nerves, can cause excruciating pain. Chest X-ray, CT chest, CT guided biopsy are the investigations (**Figs 26.7A and B**).

Pancoast Tumour

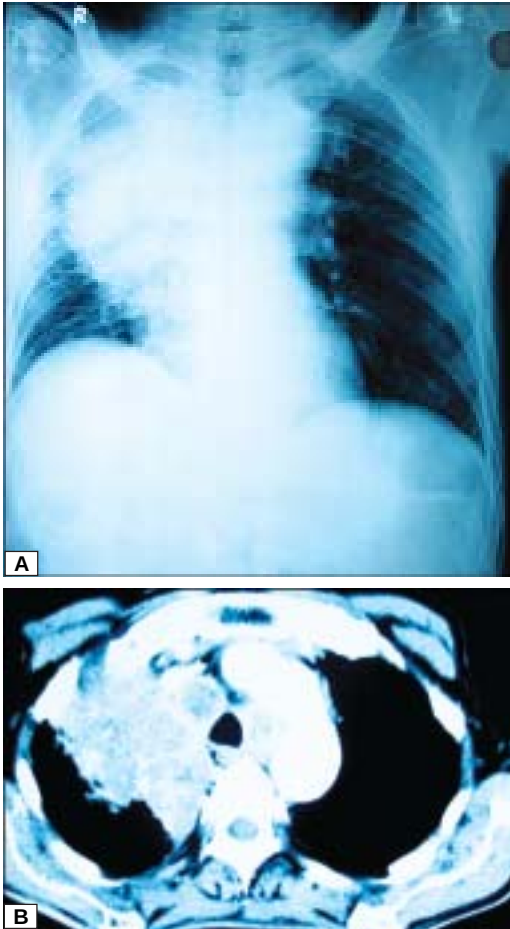
It is a type of peripheral lung carcinoma arising from the apex of the lung (5%).

Features

It invades brachial plexus, sympathetic chain, upper ribs and vertebrae causing intractable pain in upper chest and arm. CT guided biopsy is confirmative.

Pancoast syndrome (Figs 26.8A and B)

Lower brachial plexus palsy
Horner's syndrome.
Rib erosion.
Apical shadow.
Superior vena caval obstruction



Figs 26.7A and B: Chest X-ray of carcinoma lung showing chest wall involvement. CT picture showing carcinoma lung.

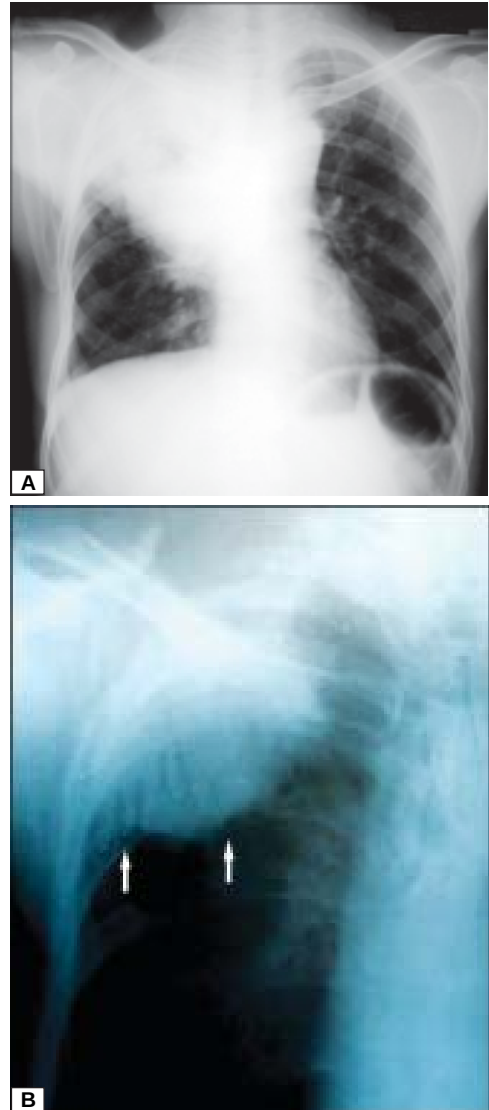
Pulmonary Complications during Postoperative Period

Precipitating Factors

Age: Common in infants and elderly.

Sex: Common in males. Common in *smokers*. and in people with chronic bronchitis, asthma, tuberculosis or *COPD*; Post-operative pain; Deep venous thrombosis and *pulmonary embolism*;

Type of surgery: Common in thoracic or upper abdomen surgeries; *Septicaemia*. Paralytic ileus. Anaesthetic complications. *Aspiration* and its problems. *Obesity*.



Figs 26.8A and B: X-rays showing right sided pancoast tumour in two different patients.

Complications

Bronchopneumonia, lung collapse, bronchitis, lung abscess, *adult respiratory distress syndrome (ARDS)*, *respiratory failure*, alkalosis, pleural effusion or empyema.

Pericarditis

Acute: Usually by bacteria like *Staph. aureus*, *H. influenzae*, *Streptococci*, *Neisseria*. It is uncommon

at present because of availability of good antibiotics. Here pus collects in the pericardial space causing decreased cardiac function and toxicity.

Chronic constrictive pericarditis (Pick's disease):

Here pericardium is thickened, fibrosed and calcified. Heart is encased in a rigid cavity which decreases the cardiac function as well as decreases the venous return.

Causes

Tuberculous pericarditis is the commonest cause; trauma; viral pericarditis; after cardiac surgery.

Clinical Features

Decreased cardiac output and tachycardia; dyspnoea and cyanosis; raised jugular veins, hepatomegaly, ascites and oedema feet.

Cardiac Tamponade

Accumulation of fluid or blood in the pericardial space causing increase in the *intrapericardial pressure* is called as *cardiac tamponade*.

Causes

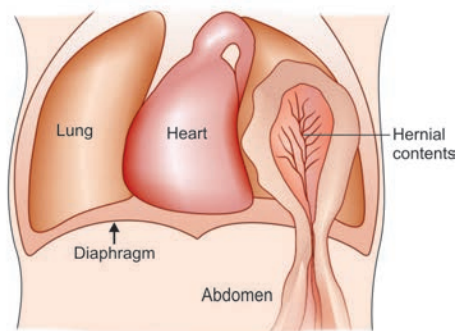
Trauma; progressive pericardial effusion due to tuberculosis, viral, bacterial infections. Often uraemia can cause significant pericardial effusion.

Clinical Features

Widened cardiac dullness and hypotension; Muffled or decreased heart sounds.; Increased venous pressure with raised jugular veins; *Pulsus paradoxus*. [pulse becomes weaker on inspiration than expiration]. In severe cases, heart is unable to expand causing *shock and often sudden death*. *Beck's triad*: Hypotension; muffled heart sounds; raised jugular venous pressure.

Diaphragmatic Hernia

It is herniation of abdominal content through diaphragm into the chest. It may be congenital or acquired (traumatic or oesophageal hiatus hernia). Congenital may be - associated with malrotation with *Ladd's band*. It may be - *Eventration* or *Hernia* through *foramen Bochdalek* or *Hernia* through *foramen*



A



Figs 26.9A and B: Diaphragmatic hernia through foramen Bochdalek. X-ray of diaphragmatic hernia left sided showing bowel loops in the left thorax.

Morgagni or Congenital oesophageal hernia (rare) (**Figs 26.9A and B**).

Types

- 1. Eventration:** It is weakening of diaphragm due to atrophy and loss of muscle of part or all of one leaf of the diaphragm, with thin fibrous tissue formation covered with pleura and peritoneum on either side. This thin diaphragm is raised higher and immobile. It is actually not a true herniation. But features mimic hernia. *Differential diagnosis* is diaphragmatic hernia through foramen Bochdalek. Condition causes respiratory embarrassment.

2. Hernia through foramen Bochdalek (through left sided pleuroperitoneal canal)

This is a developmental defective condition due to failure of fusion of pleuroperitoneal canal leaving a direct communication between pleura and peritoneum on left side. This allows herniation of contents of abdomen into the left side thorax. In 80% cases hernial sac is absent. Common content is *colon*. Occasionally small bowel and stomach are the contents. *Clinical features*: Respiratory embarrassment, scaphoid abdomen, bowel sounds in left side of chest, mediastinal shift towards right side, occasionally features of intestinal obstruction. Pulmonary hypoplasia with persistent fetal circulation is the cause and is associated with respiratory acidosis. *Investigations*: Chest X-ray, *Barium enema* (common) or Barium meal.

3. Hernia through the foramen of Morgagni: The defect lies between the sternal and costal attachments of the diaphragm and is situated in front and towards right. Colon is the commonest content. Usually it is symptom free.

4. Oesophageal hiatus hernia: Can be congenital or acquired (common).

Chest Wall Tumours

Tumours arising from the chest wall components like muscles or ribs. They can be benign or malignant. Commonest benign tumour is chondroma arising from ribs. Malignant tumours are secondaries (commonest), chondrosarcoma arising from ribs (common among primary malignant tumours), rhabdomyosarcoma from muscles, fibrosarcoma from ribs/muscles/other soft tissues, Ewing's sarcoma and invasion from other tumours like from pleura or lungs or breast.

Benign tumours are slow growing, nonmobile, painless and usually from the rib cartilage, near costochondral junction. X-ray is diagnostic, shows rib expansion with intact cortex. One or more ribs can be involved.

Primary malignant tumour has got all features of sarcoma—progressive rapid enlargement, attaining large size, warm, vascular, nonmobile, often extends into the thoracic cavity or with skin ulceration. Secondaries in *lung/brain/liver* can occur. Chest X-ray, CT chest, US abdomen should be done to see secondaries. CT scan can also give idea about the tumour extension and operability. Open incision/trucut biopsy is essential for histological confirmation (**Fig. 26.10**).



Fig. 26.10: Chest wall tumour – over the sternum – could be secondary or primary chondrosarcoma.

Examination of Hand and Foot

EXAMINATION OF HAND

History

Following points to be noted—

- History of trauma
- History of swelling, onset, progress, pain.
- History of deformity and loss of function.
- Family history related to the condition.

Examination

Inspection

Following aspects to be noted—

- *Attitude* of the hand—always compare to opposite side.
- *Swelling*—redness, localised or diffuse, size, shape and extent.
- *Wasting* of thenar and hypothenar eminence.
- Presence of ulcers, gangrene over the finger tips.
- Changes in the forearm and arm.
- Inspection of the axilla.

Palpation

There is local rise of *temperature* in hand infections—superficial or deep; in the joints. Fingers will be cold in ischaemic conditions like cervical rib syndrome.

Tenderness may be present over the swelling and other areas. Specific point tenderness is typical in certain type of hand infections. *Kanavel sign* is typical of suppurative tenosynovitis.

Pulses should be checked. Radial and ulnar pulses should be checked. Allen's test should be done.

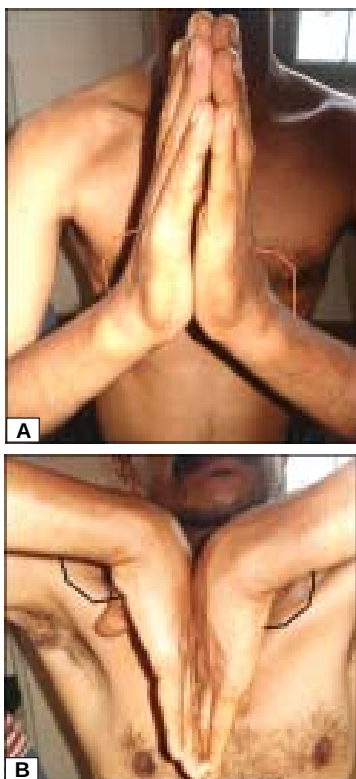
Sensations: There may be hyperaesthesia, paraesthesia (tingling, numbness, pins and needles sensation),

decreased sensation (blunting). Anaesthesia (absence of sensation) should be checked. Cotton, needle, sense of position, tuning forks, and cold and warm water tubes can be used to check various sensations. Sensory distribution in hand should be checked properly. Sensations in the distribution of radial, ulnar and median nerves should be checked (Refer Chapters 8: Examination of Peripheral Nervous System and 9: Examination of Muscles, Tendons and Fasciae).

Movements of the fingers and wrist joint should be checked. Flexion (midcarpal joint), extension (radiocarpal joint), abduction (midcarpal joint), adduction (radiocarpal joint) and circumduction (flexion → adduction → abduction → extension) should be checked. Thumb shows flexion, extension, abduction, adduction and opposition movements at carpometacarpal joint. Fingers at metacarpophalangeal joints show flexion, extension, abduction and adduction. Flexion and extension are the only movements in interphalangeal joints of fingers. Both *active* (by the patient) and *passive* (by the examiner) movements should be checked. Flexion is 60°; extension is 70°; adduction is 35°; abduction is 25°. Extension is checked by Indian method of salutation by keeping both hands in contact. Flexion is checked by placing backs of hands in contact. Pronation and supination takes place at inferior radioulnar joint. In wrist arthritis *all movements* are painful and restricted (**Fig. 27.1**).

Axillary nodes should be examined.

Neck should be examined for cervical rib, thrill, and bruit. Cervical spine should be examined. Neck movements should be checked.



Figs 27.1A and B: Extension (radiocarpal) and flexion (midcarpal) of wrist.

Modified Verdan zone system in the hand (Tendon zones) (Fig. 27.2)

Zone I

From the fingertip upto the attachment of flexor digitorum superficialis (Middle of middle phalanx). It contains Flexor digitorum profundus.

Zone II

It begins proximal to metacarpophalangeal joint at distal palmar crease and extends distally up to the attachment of flexor digitorum superficialis at the middle of the middle phalanx. It is called as '**No-man's-land**'. Here flexors are tightly enclosed within a fibro-osseous tunnel. It is the **most dangerous zone** in hand injuries (**Critical zone**).

Zone III

It begins at the distal end of flexor retinaculum (base of the palm) and ends at the transverse crease of the palm. It contains lumbricals attached to flexor digitorum profundus.

Zone IV

It begins at the proximal end and ends at the distal end of flexor retinaculum (**Fig. 27.3**).

Zone V

It extends from the proximal end of flexor retinaculum proximally upto distal third of the forearm.

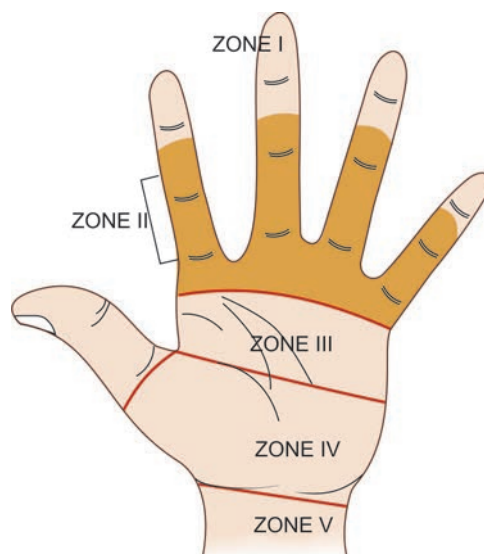


Fig. 27.2: Modified Verdan zone system in the hand.

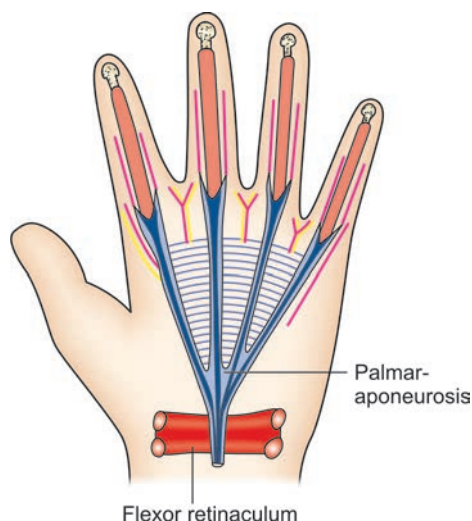


Fig. 27.3: Flexor retinaculum.

Hand Infections

Hand is a compact actively functioning unit. It contains neurovascular bundles, muscles, bones and ligaments. Infection can occur due to minor injuries, or by haematogenous spread.

Precipitating causes: Diabetes; Immunosuppression; Trauma; HIV infection; Steroid therapy.



Fig. 27.4: Subcuticular infection with pus formation.

Common organisms: *Staphylococcus*, *Streptococcus*, Gram negative organisms like *E. coli*, *Klebsiella*, *Pseudomonas*. Occasionally fungal infection causes chronic paronychia, Madura hand due to *Nocardia* group of fungi, viral infection like ORF (*Parapox virus infection causing contagious pustular dermatitis of hand*) can occur (Fig. 27.4).

General Features of Hand Infection

Infection spreads faster in all areas. It causes oedema over the dorsum of hand due to lax skin and more lymphatic network even though infection per se is more over the volar aspect. It looks like **frog hand**. There are restricted movements of fingers and hand. The hand functions like **hook, pinch, grip, grasp** are lost. Severe pain and tenderness, with fever are other features. Tender palpable axillary lymph nodes are often present.

Different Types of Hand Infections

Acute paronychia; Chronic paronychia; Terminal pulp space infection (**felon**); Subungual infection; Web space infection; Mid palmar space infection.; Thenar space infection; Deep palmar abscess; Acute suppurative tenosynovitis; Chronic tenosynovitis of flexor tendon sheath of palm and forearm – Compound palmar ganglion. Lymphangitis of the hand.

Investigations

Pus for culture and sensitivity; blood sugar; urine sugar and ketone bodies; X-ray of the part.

Complications of hand infections

Stiffness of digits and hand (ankylosis)
Deformity and disability
Bacteraemia and septicaemia
Osteomyelitis of bones depending on location of abscess like metacarpal bones, terminal phalanx
Suppurative arthritis of joints
Paralysis of median nerve

Acute Paronychia

It is the most common hand infection. It occurs in subcuticular area under the *eponychium*. Minor injury to finger is the common cause. Suppuration occurs very rapidly. It tracks around the skin margin and spreads under the nail causing *hang nail* or *floating nail*. Organisms are *Staphylococcus aureus* and *Streptococcus pyogenes*.

Clinical Features

Severe throbbing pain and tenderness (dependent throbbing) with visible pus under the nail root. Nail on touch is very tender (paronychia means 'Run around') (Figs 27.5A and B).



Figs 27.5A and B: Paronychia – early stage in one patient; with pus formation in other.

Chronic Paronychia

It is commonly due to fungal infection.

Clinical Features

Itching in the nail bed; recurrent pain; discharge; secondary bacterial infection may supervene.

Terminal Pulp Space Infection (Felon)

It is the second most common hand infection (25%). Index and thumb are commonly affected. Usually occurs by a minor injury like finger prick.

Surgical Anatomy

Terminal pulp space contains fat and is partitioned by septae which attaches periosteum of terminal phalanx to skin. Proximally deep fascia is attached to the periosteum distal to the base of terminal phalanx, i.e. distal to the attachment of flexor tendon. So, terminal space is a closed compartment which causes increased pressure when there is infection, compresses terminal artery leading to thrombosis, resulting in osteomyelitis of terminal phalanx (**Fig. 27.6**).

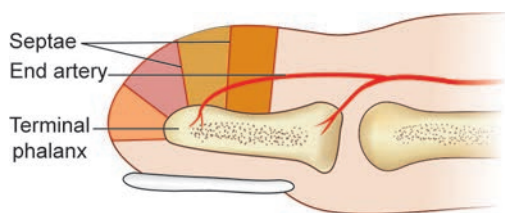


Fig. 27.6: Anatomy of the terminal pulp space.

Bacteria

Staphylococcus, *Streptococcus*, Gram negative organisms.

Clinical Features

Presents with pain, tenderness, swelling in the terminal phalanx, fever, and tender axillary lymph nodes. Often suppuration is severe, forming *collar stud abscess* which eventually may burst.

Complications

Osteomyelitis of the terminal phalanx.

Infection of Webspaces

Surgical Anatomy

There are three triangular web spaces filled with fat between the dorsal and volar skin. When space is filled with pus it straddles the deep transverse ligament. Even though pus is volar, it points out dorsally. It originates

from—abrasion; infection of proximal volar space of finger, callosities, infection of proximal spaces.

Bacteria

Staphylococcus, *Streptococcus*, Gram negative organisms.

Clinical Features

Present with fever; pain and tenderness; oedema of dorsum of hand; *Maximum tenderness* is on the volar aspect; *V sign*—separation of fingers. If untreated infection may spread into other web spaces and hand spaces.

Deep Palmar Space Infection

Surgical Anatomy

Two deep palmar spaces are present - *midpalmar space*; *thenar space*. Midpalmar space is bounded in front by palmar aponeurosis, behind by medial three metacarpals, laterally by a vertical line from lateral margin of the middle finger. It contains flexor tendons, neurovascular bundles and lumbricals. It is the common site of the infection. Thenar space is located anterior to lateral two metacarpals. Infection here is usually due to extension from midpalmar space.

Causes

Trauma, spread from infection of finger spaces and web spaces, haematogenous spread, spread from tenosynovitis.

Clinical Features

Present with pain and tenderness in the palm; oedema of dorsum of hand (*frog hand*), loss of concavity of palm, painful movement of metacarpophalangeal joint (but interphalangeal joint movements are normal and painfree), fever; palpable tender axillary lymph nodes. Eventually pus may come out of palmar aponeurosis forming collar stud abscess and later sinus formation.

Complications

Osteomyelitis of metacarpals, stiffness of hand, suppurative arthritis, extension of infection into other spaces.

Acute Suppurative Tenosynovitis

It is the bacterial infection of flexor tendon sheaths.

Surgical Anatomy

Radial bursa is flexor sheath of flexor tendon of thumb which extends to the digit. **Ulnar bursa** is flexor sheaths of medial four flexor tendons which extend into the digit of the fifth [little] finger (**Fig. 27.7**).

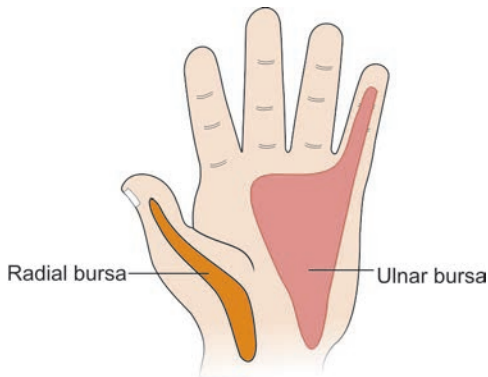


Fig. 27.7: Bursae (Flexor) in hand.

Common Bacteria

Staphylococcus aureus, *Streptococcus pyogenes*.

Clinical Features

Presents with symmetrical swelling of entire finger; Flexion of finger—*Hook sign*; Severe pain on extension; tenderness over the sheath; oedema of whole hand, both palmar and dorsal aspect (due to lymphatic spread). In case of ulnar bursa infection, as it is extending into the little finger, pain and tenderness extends into little finger but not much to other fingers. In radial bursa infection, thumb is swollen with pain and tenderness over the sheath of the flexor pollicis longus and there is inextensibility of interphalangeal

joint. Swelling just above the flexor retinaculum is common.

Complications

Spread of infection proximally into forearm; stiffness of fingers and hand; suppurative arthritis; osteomyelitis; median nerve palsy; bacteraemia and septicaemia.

Parona's space is space in the forearm to which infection can occur as extension from the hand.

Apical Subungual Infection

It is infection of the space between subungual epithelium and the periosteum. It occurs after minor trauma or rarely after formation of subungual haematoma. Beneath the free edge of the nail, pus comes to the surface. Excruciating tenderness with small visible pus under the tip (summit) of the nail is the feature. Drainage with 'V' incision over the summit is the treatment along with antibiotics. Osteomyelitis is not common (**Figs 27.8A and B and 27.9**).

Compound Palmar Ganglion

It is chronic tenosynovitis of flexor tendon sheaths due to tuberculosis (tuberculous tenosynovitis) or rheumatoid arthritis. It can be unilateral or bilateral.

Pathology

Flexor tendon sheath on either side of the wrist (Flexor retinaculum) is involved, i.e. both in the volar surface of palm and lower forearm. Swelling contains fluid with typical **melon seed bodies**. Condition is often bilateral in case of rheumatoid arthritis.

Clinical Features

Swelling in the palm and lower forearm which is smooth, soft, nontender, fluctuant and cross fluctuant across flexor retinaculum, and transilluminant. Wasting

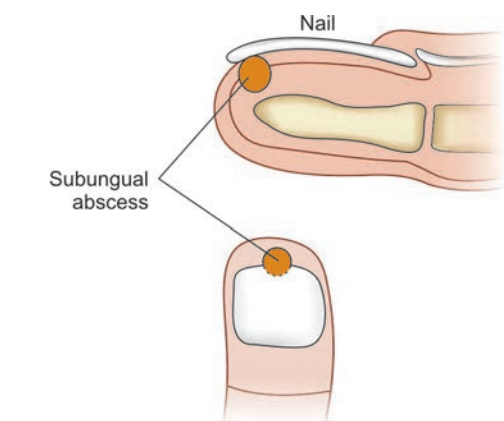
In HAND

Do's

- Do examine hand carefully.
- Do think of other diagnosis.
- Do wait for abscess to localize.
- Do place adequate length and depth of incisions.
- Do immobilize, elevate the hand.
- Do give antibiotics and proper dressings.

Don't's

- Do not incise every infected digit.
- Do not make puncture incisions or over pads.
- Do not injure the digital nerves or vessels.
- Do not place incisions crossing the crease line.
- Do not close human bites or lacerated wounds.
- Do not forget to send pus for culture and sensitivity.



A



B

Figs 27.8A and B: Apical subungual infection and pus formation.



Fig. 27.9: Subungual haematoma.

of hand and forearm muscles can occur. Matted axillary lymph nodes may be palpable. Primary focus may be present in lungs.

Spina Ventosa

It is phalangeal tuberculosis (*Tuberculous dactylitis*). It is called as spina ventosa because of its appearance as 'Air filled balloon' (**Fig. 27.10**).



Fig. 27.10: Spina ventosa – tuberculous dactylitis.

Milker's Nodes

Milker's nodes is similar viral infection of the hand seen in cow handlers.

Hand Injuries

Classifications

Tidy injuries: They are clean incised wounds and are usually treated by primary suturing and also depends on the tissues involved like nerves, tendons and muscles.

Untidy injuries: They are lacerated wounds and treated by debridement and later by delayed primary or secondary suturing.

Compartment injuries.

Degloving injuries

Indetermined injuries which could not be assessed.

Assessment of Injury

Should include—number, extent, depth, deformity—disability, neurovascular injuries, tendon injuries, muscle injuries bone and joint injuries (**Figs 27.11A to C and 27.12**).



Figs 27.11A to C: Different types of hand injuries—abrasions, deep wound with loss of fingers, extensive crush injury.



Fig. 27.12: Ring causing block and distal oedema due to vascular compression.

Syndactyly

It is *webbing or fusion* of fingers.

Causes

Congenital and hereditary – Common; Traumatic like burns (**Fig. 27.13**).

Types

Cutaneous; Fibrous; Bony—complex. *Features:* It can be unilateral or bilateral. Often there will be webbing of toes also. If bony type is suspected X-ray of the part should be taken.



Fig. 27.13 Syndactyly.

Remember

Hand should be flexible and strong; sensitive and pain free and coordinated to show all fine and powerful functions. *Pinch* (picking a small object); *power grip* (holding a hammer); *key grip* (holding a key); *chuck grip* (holding a pen); *hook grip* (carrying a bag) – are the functions of hand.

Hand should be properly examined clinically for tendon functions; neurological problems – sensations (sweat test, two point discrimination test); for circulation (Allen's test); joint movements; examination of entire upper limb; opposite hand; axillary lymph nodes and relevant systemic examinations.

Nerve conduction studies; electrophysiology; MRI hand; radioisotope bone scan; selective angiograms; X-ray hand are the relevant investigations other than systemic investigations.

Elevation to reduce oedema; splintage to prevent contracture; early movements once inflammation subsides; early exploration of wound or surgical drainage of infective area; regional anaesthesia; usage of tourniquet; incisions when cross the flexor creases, it should be at 45° angle are the principles of treatment.

EXAMINATION OF FOOT

History

History of pain is asked. Location of pain, whether over 2nd metatarsal bone or between 3rd and 4th metatarsals has to be noted. Pain in the heel may be due to plantar fasciitis. Type, severity of deformity should be asked. Whether it interferes with patient's walk or not is noted.

Examination

Inspection

Inspection is done in standing; both feet should be inspected. Affected leg is inspected for deformity, position, wasting, ulcers, proximal parts of the limb, etc. Ulcer may be trophic (**Fig. 27.14**).

Palpation

It is done for swelling and ulcer. In detail each of these should be examined (**Figs 27.15A and B**).

Movements and Gait

Movements occurring at ankle joint (dorsiflexion and plantar flexion) and subtalar joint (inversion and eversion) should be checked. In *dorsiflexion*, front of leg is approximated to dorsum of foot with reduction in the angle between two. In *plantar flexion* toes point downwards; heel is raised with increased angle between foot and leg in front. *Inversion* takes place in subtalar joint with plantar aspect of foot facing medially; in *eversion* plantar aspect of the foot faces laterally. Dorsiflexion is by tibialis anterior; plantar flexion is



Fig. 27.14: Polydactyly foot—two great toes.

by gastrocnemius and soleus. Inversion is by tibialis anterior and tibialis posterior. Eversion is by peroneus longus and peroneus brevis.

Muscle Power, Sensations, Reflexes and Gait

Proper neurological examination should be done.

Examination of Knee, Hip and Spine

Knee, hip and spine should be examined. Often spina bifida is an association of talipes equinovarus. Genu valgum, coxa vara is often associated with deformity of foot.



Figs 27.15A and B: Different types of infections in foot – subcuticular abscess, cellulitis, abscess. Often it may cause osteomyelitis of metatarsals and gangrene.

Examination of Inguinal Lymph Nodes

Proper examination of inguinal lymph nodes is to be done.

Investigations

X-ray foot; MRI foot; X-ray spine (if needed) are the needed investigations.

Flat Foot/Pes Planus (Fig. 27.16)

It is flattening of longitudinal arches of the foot causing flattening of the normal concavity on the inner aspect of the foot with prominent navicular bone; limitations of movements of tarsal joints; tender spring (inferior calcaneonavicular) ligament. *Causes* – genu valgum, outer rotation of tibia, forefoot varus, congenital, infantile, obesity, postural (weak intrinsic foot muscles), spasmodic (peroneal muscles), chronic illness.

Pes Cavus

It is also called as *hollow foot*, with claw toes and high arch. Present with thick splayed forefoot; weak intrinsic muscles; prominent metatarsal heads; callosities with osteoarthritis of tarsal joints. It can



Fig. 27.16: Flat foot.

be familial, may be associated with spina bifida; may be due to poliomyelitis; or idiopathic (commonest).

Talipes Equinovarus/Club Foot

It is usually congenital; common in boys; commonly bilateral; foot is turned inwards with sole directed

medially; with poor development of calf muscles. Components are – inversion of foot; adduction and inward deviation of forefoot; plantar flexion/equines. There is subluxation of talonavicular joint with navicular bone lying more medially. Acquired variety is seen in infantile paralysis and is unilateral.

Talipes calcaneovalgus is rare congenital deformity. It is opposite of talipes equinovarus with foot being everted and dorsiflexed.

Talipes

Talipes equinus—walk on toes.

Talipes calcaneus—walk on heel.

Talipes varus—walk on lateral margin with sole looking medially.

Talipes valgus—walk on medial margin with sole facing laterally.

Talipes equinovarus—inverted plantar flexed foot.

Talipes calcaneovalgus—everted dorsiflexed foot.

Ingrowing Toe Nail (Onychocryptosis)

It is also called as *embedded toe nail*. It is due to inward curling of side of the nail causing it to form a lateral spike which results in repeated irritation and infection of overhanging tissues in the nail fold.

Causes

Tight shoes; Improper cutting of nails (very short and convexly).

Clinical Features

It is common in **great toe** and is often **bilateral**. Both medial and lateral (common) sides of the toe can be involved. Pain, tenderness, swelling of margins of the toe, often with foul smelling discharge are the other features (**Figs 27.17A and B**).



Figs 27.17A and B: Ingrowing toenail.

Onychogryphosis (Greek-hooked Nail)

It is curving of nail upwards (**Ram's Horn Nail**). Thickened, curved, over grown nail looks like a ox horn. It is seen in bed ridden people, may be due to fungal infection.

Onychomycosis

It is fungal infection of the nail of great toe; nail becomes brittle, discoloured and splits longitudinally.

Athlete's Foot

It is the fungal infection of the skin between the toes—*Tinea pedis*. Fungi enter through cracks; survive due to moisture in between toes. Skin is swollen, red, with sticky fluid, macerated with blisters. Itching, deep cracks, pain and discharge are common features.

Hallux Valgus

Here great toe is deviated laterally at metatarsophalangeal joint. It is common in females. It may be due to persistent lateral force or occasionally hereditary. There is outward deviation of big toe with medial deviation of first metatarsal head. Thick walled bursa (*bunion*) over medial aspect of the head of the first metatarsal bone and osteoarthritis of the metatarsophalangeal joint are the eventual complications. It is often associated with *hammer toe* (**Fig. 27.18**).



Fig. 27.18: Hallux valgus.

Hammer Toe

It is fixed flexion deformity of interphalangeal joint – usually of proximal interphalangeal joint of 2nd toe with compensatory hyperextension of the distal interphalangeal joint with an overlying callosity.

Hallus Rigidus

It is osteoarthritis of metatarsophalangeal joint of great toe with osteophytes in the margin, with a bunion / bursa over it. Pain, restricted joint movement, forced dorsiflexion are the features. Narrowed joint space with sclerotic bones and osteophytes is typical.

Morton's Metatarsalgia

It is fibrous thickening or neuroma of 3rd digital nerve of foot just proximal to its division between 3rd and 4th space. It is common in middle aged female; pain in the 3rd interdigital space which radiates on the sides of the 3rd and 4th toes with localised tenderness and often swelling. It is plantar digital neuritis.

Stress/March/Fatigue Metatarsal Fracture

It is hairline fracture of 2nd or 3rd metatarsal bone near its neck which later during spontaneous healing forms a large callous around the fracture site mimicking tumour. It begins after unusual long walk or marching causing pain and tenderness in the forefoot.

Sever's Disease

It is calcaneal apophysitis seen in children at the attachment of posterior apophysis. It was earlier thought to be osteochondritis.

Causes of painful heel

- Calcaneal diseases
- Subtalar arthritis
- Calcaneal tendon rupture
- Calcaneal paratendinitis
- Calcaneal bursitis
- Calcaneal apophysitis
- Tender heel pad
- Calcaneal spur, plantar fasciitis
- Osteochondritis of bones
- Morton's metatarsalgia
- March fracture
- Hallux valgus / rigidus / hammer toe

Freiberg's Disease

It is partial necrosis and fragmentation of usually 2nd or 3rd metatarsal head which gets deformed eventually developing osteoarthritis of adjacent joint. It could be metatarsal *osteochondritis dissecans*. It is seen in adolescent girls.

Kohler's Disease

It is osteochondritis of navicular bone which becomes denser; presents in child < 5 years with pain, limp, thickening of the navicular bone.

Examination in Head, Chest and Abdomen Injuries

EXAMINATION IN HEAD INJURIES

History

Detailed history of an accident should be taken from the attender as patient may be unconscious or may be in shock and so unable to narrate the history. Type of trauma – road traffic accident, assault, fall from height is asked. Slipping and falling, etc. may also be the mode of injury.

History of vomiting is important as it may indicate that there is intracranial injury. It is usually repetitive and projectile. Vomitus may contain blood in fracture of middle cranial fossa. Often it may be a sign of recovery also.

History of convulsion suggests intracranial injury. It may be due to haemorrhage or cerebral injury. Time, type, duration of convulsion should be asked. In middle meningeal haemorrhage it is unilateral (*Jacksonian*) starting from toes going upwards towards face.

Level of consciousness: Patient may be conscious initially but later becomes unconscious. How long after the trauma, patient remains unconscious should be assessed. Duration of unconsciousness, recovery should be asked. Patient may become unconscious initially then recovers soon and after certain period of time becomes unconscious again. In the interval patient behaves normally. This time interval of relative conscious period is called as '*lucid interval*'. It is usually seen in extradural haemorrhage.

Post-traumatic amnesia (PTA): It is to be noted that how long after trauma, patient was suffering from loss of memory. It is confirmed by patient's attender. **Grading of PTA:** *Grade 1* (slight) – less than 1 hour;

Grade 2 (moderate) 1- 24 hours; *Grade 3* (severe) is 1-7 days; *Grade 4* (fatal) is more than 7 days **Headache:** It is important sign in head injury. It suggests slowly progressive haematoma inside.

Swelling in the scalp may suggest scalp haematoma with a fracture underneath. It may be progressive also.

Bleeding from the nose, ear, and mouth suggests injury to the base of skull. Watery discharge from these places suggest CSF leak.

Other earlier history of blood pressure, diabetes, history of smoking and alcohol intake, intake of drugs and sedatives, history of epilepsy or receiving any other treatment are to be noted. Family history of epilepsy is also important.

Examination

General Examination

Pulse (tachycardia, *bradycardia*), respiration, presence of shock has to be noted; other system should be examined for associated injuries which may be of priority in such occasions (abdomen bleed, haemothorax). Tachycardia is seen in cerebral concussion, slow bounding pulse with hypertension is a feature of cerebral compression to maintain cerebral circulation. Irregular pulse suggests poor prognosis.

Temperature: Hyperpyrexia is common in pontine haemorrhage, intraventricular haemorrhage and brain stem injury. Initially in cerebral concussion temperature may be low. In brain compression it raises. Temperature may be more by 1-2°F on the paralysed side – *Victor Horsley's sign*. Systolic hypertension is common.

Respiration: It is slow and deep in compression; shallow in concussion. Blowing of lips and cheeks with each breathing; relaxed soft palate and tongue fall causing airway obstruction with snoring suggests compression of medulla suggesting poor prognosis. *Cheyne Stokes respiration* suggests poor prognosis. Crepitations and altered breath sounds may suggest aspiration often with pneumonia.

Local Examination

Scalp

Scalp haematoma may be subcutaneous (superficial, mobile), subaponeurotic (extends beyond skull bone suture lines), subpericranial (limited to one cranial bone by suture line).

Wound: Superficial/deep, deep with gaping if galea is injured. Its extent, number, depth, presence deeper bone fracture – should be checked. Boggy swelling in the temporal region may suggest temporal bone fracture often with extradural haematoma. Ecchymoses appearing near the tip of the mastoid process in 3–4 days (*Battle's sign*) suggests posterior cranial fossa fracture behind the foramen magnum.

Position

Fully unconscious patient will lie flaccid and relaxed. May remain curled to one side in cerebral irritation; Restless with changing positions are also common.

Level and Depth of Consciousness

It should be checked according to coma scale. Patient's response is checked by pressing over the glabella and looking for facial expression. Absence of corneal reflex and urinary/faecal incontinence are significant. Grading of response to pain stimulus – *Grade I* – Avoid and push the stimulus with attempt; *Grade II* – Simple grunt; *Grade III* – Reflex decerebrating posture.

Bleeding from Nose, Ear and Mouth

Bleeding from nose suggests fracture of anterior cranial fossa; bleeding from mouth and ear suggests fracture in middle cranial fossa; CSF leak along with blood makes it watery. Facial palsy, deafness, meningitis can develop. Occipital bone sinuses may get torn in fracture

of posterior cranial fossa causing dilated nonreactive pupil, Cheyne-Stokes breathing.

Neurological Deficits

One should check for sensation if patient is conscious; muscle power, rigid or flaccid paralysis; plantar reflex (Babinski's sign), abdominal reflexes. Joint jerks (Knee, ankle, biceps, and triceps) should be checked. Reflexes are useful in unconscious patient to identify paralysis. Complete neurological examination should be done.

Neck rigidity is an important sign to be looked for. It suggests subarachnoid haemorrhage, fracture dislocation of cervical spine or meningismus or meningitis.

Ataxia, nystagmus are features of cerebellar injury.

Cranial Nerve Examination

All cranial nerves should be examined (See chapter 15). Oculomotor nerve is most important. During initial period of cerebral compression nerve gets irritated causing constriction of same side of pupil. Eventually nerve paralysis occur causing pupil dilatation.

Examination of Eye

In *fracture of anterior cranial fossa* extravasation of blood and ecchymoses occurs after 24 hours first at lower (by gravity) than at upper eyelid – limiting to orbital margin. *Sub conjunctival* haemorrhage (is deep to conjunctiva and does not move with conjunctiva) pointing towards the cornea with invisible posterior limit with conjunctival oedema can occur (**Figs 28.1A and B**). Eyeball may be pushed forward with limitation of movements.

'*Black eye*' is due to local injury around orbit with appearance of ecchymoses early/immediately which spread beyond orbital margin into the cheek, nose and forehead. *Superficial conjunctival haemorrhage* is confirmed by its mobility with the conjunctiva and visibility of its posterior limit.

Pupils in cerebral concussion are equal, react to light and may be slightly dilated. *Hutchinson's pupil* is a feature of progressive cerebral compression showing three stages – *Stage I* – irritation of oculomotor nerve causing constriction of the pupil on injured side with normal sized pupil on opposite side. *Stage II* Oculomotor nerve paralysis causes dilatation of pupil



Figs 28.1A and B: Black eye and subconjunctival haemorrhage.

on injured side with irritation of opposite oculomotor nerve causing constriction of pupil on opposite side. *Stage III* – both side pupils are dilated and fixed, not reacting to light.

Pin point pupil (fixed); pyrexia; paralysis are the (triad) features of *pontine* haemorrhage.

Examinations of Other Systems

It is mandatory to examine chest, abdomen and limbs. Often life threatening injury and haemorrhages in the abdomen and thorax may be missed or ignored and patient may succumb to that. If there are such injuries they take priority as otherwise haemorrhage may be life threatening.

Reassessment of the patient by repeated examinations is needed.

Investigations

Haemoglobin, PCV, electrolytes, blood gas analysis depending on the clinical requirement.

CT scan is ideal investigation to identify intracranial injuries. Usually plain CT is done in emergency situation (**Fig. 28.2**).



Fig. 28.2: Intracerebral haematoma – CT picture.

Carotid angiography is done to see the site of bleeding and displacement of cranial vessels.

MRI is very useful and sensitive.

Mechanism of Head Injury

Distortion of the brain: Brain is a soft structure, therefore has 'mobility' and readily distorts. This distortion and mobility is accentuated by CSF and vascular components. Any impact creates shearing forces in the brain causing damage to neurons, supporting tissues and blood vessels. This leads to loss of consciousness, focal neurological deficits. Such distortive damage may be temporary or permanent. **Mobility of the brain in relation to the skull and membranes** causes cerebral damage and bleeding in dural spaces from torn vessels in the dura, commonly the veins. In old age, the brain shrinks, as a result of which 'mobility' of brain increases favouring rupture of veins which cross the subdural space.

Configuration of interior of skull: Damage is less severe over the smooth area but is more severe over the rough and sharp areas. So the damage is severe over the anterior cranial fossa, over the falx, and over the tentorium.

Deceleration and acceleration injuries: *Deceleration injuries* occur when moving head strikes an immovable object (like in road traffic accidents). *Acceleration injuries* occur when stationary skull is struck by a moving object (like in assault).

Cerebral concussion is slight distortion causing temporary physiological changes leading to transient loss of consciousness with complete recovery. *Cerebral contusion* is more severe degree of damage with bruising and cerebral oedema leading to diffuse or localized changes. *Cerebral laceration* is tearing of brain surface with collection of blood in different spaces and with displacement of dural parts.

Effects of Brain Injuries

Brain oedema is accumulation of fluid both intracellular and extracellular. It is due to congestion and dilatation of blood vessels. It may be diffuse or localized.

Brain necrosis is of severe variety with destruction and is due to haemorrhagic infarction.

Extradural haematomas occurs usually in temporo-parietal region. It is commonly due to tear in middle meningeal veins and often middle meningeal artery. It causes intracranial hypertension, displacement, *Kernohan's effect* and often death. *Subdural haematoma* is due to tear of veins between cerebrum and dura due to shearing forces. It is diffuse and commonly associated with cerebral injury. *Intracerebral haematoma* can occur in different parts of cerebrum may

be in frontal lobe, temporal lobe. *Intraventricular haemorrhage* is very severe type of haemorrhage.

Brain ischaemia is due to increased pressure. This in turn leads to alteration in the perfusion of brain which itself aggravates the ischaemia and this forms a vicious cycle, causing progressive diffuse ischaemia of brain.

Coup injury occurs on the side of the blow to the head. **Contre-coup** injury occurs on the side opposite to the blow on the head. **Coning:** It is due to rise in intracranial pressure causing either—Herniation of contents of supratentorial compartment through the *tentorial hiatus* **or** Herniation of the contents of infratentorial compartment through the *foramen magnum*. In supratentorial herniation, there is compression of ipsilateral third cranial nerve and midbrain. Midbrain is displaced away from the mass (haematoma) and midbrain is pressed by the sharp edge of tentorium cerebelli of opposite side leading to dysfunction of corticospinal fibres (which after decussation supplies on the opposite side in the body, i.e. same side of the injury). This leads to: deterioration in the level of consciousness; dilatation of pupil on the side of compressing mass (haematoma); hemiparesis on the same side of the mass lesion (haematoma) due to compression of the contralateral corticospinal tract. This effect is called as **Kernohan's notch (Fig. 28.3)**. Herniation of infratentorial contents through the foramen magnum causes obstruction of cerebral aqueduct which further damages the brain function.

GLASGOW COMA SCALE

Eye opening		Verbal response		Motor response	
Spontaneous.	4	Oriented.	5	Obeys commands.	6.
To Speech.	3	Confused.	4	Localises pain.	5.
To Pain.	2	Inappropriate words.	3	Flexion to pain.	4.
None.	1	Incomprehensible words.	2	Abnormal flexion.	3.
		None.	1	Extension to pain.	2.
				None.	1.
Total score—15.					
Mild head injury: score 13-15.					
Moderate head injury: 9-12.					
Severe head injury: less than 8. (3-8).					
Score 1—dead or dying. Score 2—vegetative state. Score 3—severe disability.					
Score 4—moderate disability. Score 5 —good recovery.					

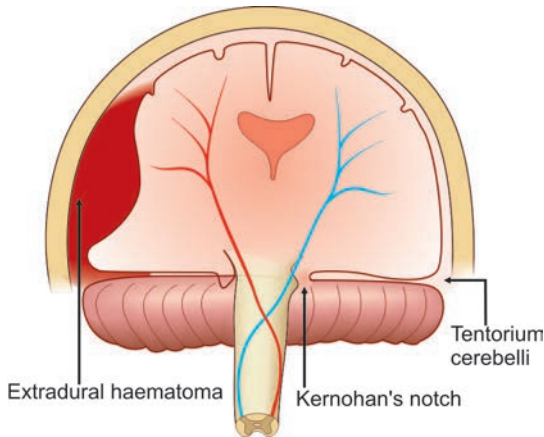


Fig. 28.3: Coning with Kernohan's notch.

Respiratory failure is due to altered pO_2 , pCO_2 levels.

Raised intracranial pressure causes bradycardia, hypertension, vomiting. Raised intracranial pressure may precipitate coning and thus aggravates brain ischaemia.

Hyperpyrexia, convulsions is seen due to irritation of grey matter.

CSF rhinorrhoea or CSF otorrhoea. Fluid and electrolyte imbalance are other features.

Adelaide Coma Scale

It is used in children. Scores for eye opening and motor responses are same as Glasgow coma scale. But *verbal response score differs*—Oriented-5. Words-4. Vocal sounds-3. Cries-2. Nil-1. *Orientation cannot be evaluated below 5 years. For first 6 months, the best verbal response is CRY.*

Indications for Admission

Any altered level of consciousness; Skull fracture; Focal neurological features; Persistent headache, vomiting, systolic hypertension, bradycardia; Alcohol intoxication; Bleeding from ear or nose; Associated injuries.

Complications of Head Injuries

Early: (1) Brainstem injury—due to coning. (2) Compression over cerebellum and medulla. (3) **CSF rhinorrhoea:** It is due to communication between intra-

cranial cavity and the nose. There is a tear in the dura following the fracture involving the sinuses - frontal, ethmoid, sphenoid sinuses. Meningitis is the common complication of CSF rhinorrhoea. (4) Meningitis - common. (5) Pituitary damage and endocrine failure - requires high dose of hydrocortisone 200 mg 6th hourly. (6) Aerocele. (7) CSF otorrhoea. (8) Depressed fractures will often cause injury to dural venous sinuses and may lead to torrential haemorrhage which may be life threatening. So such depressed fractures should never be elevated.

Late: Chronic subdural haematoma; Early post-traumatic epilepsy—they need anticonvulsants for 3 years; Late post-traumatic epilepsy is due to scarring and gliosis of cerebrum; Post-traumatic amnesia. Post-traumatic hydrocephalous; Post-traumatic headache.

Extradural Haematoma

It is collection of blood in the extradural space between the dura and skull. Commonest site is *temporo-parietal* region. It can be *unilateral or bilateral*. Vessels commonly involved: Middle meningeal veins; Anterior branch of middle meningeal artery; Posterior branch of middle meningeal artery. Usually is associated with fracture of temporo-parietal region.

Pathology

Direct blow like from cricket ball or road traffic accidents or fall and impact or coup and contre coup injuries → Fracture of thin temporal bone → tear of vessels → bleeding initially outward towards the scalp and under temporalis muscle → formation of haematoma → gradual stripping of dura from skull and collection of blood occurs → in 6-12 hours extradural haematoma occurs which raises the intracranial pressure → **Coning** of supratentorial content (uncus of temporal lobe) through tentorial hiatus → shift of midbrain towards opposite side which gets injured by sharp edge of the tentorial cerebelli → corticospinal tract before decussation on opposite side gets injured → *So hemiparesis, and pupillary changes occurs on the same side of haematoma* → this effect is called as **Kernohan's notch effect**. Immediately after injury, there will be transient loss of consciousness and the patient soon becomes normal. Later after 6-12 hours, he again falls ill and the condition deteriorates. This

is the time taken to develop raised intracranial pressure, coning and its effects. This crucial time gap which is unnoticed and often missed is called as '**lucid interval**'.

Clinical Features

History of transient loss of consciousness following a H/O blow or fall. Patient soon regains consciousness and again after 6-12 hrs starts deteriorating (**Lucid interval**). Later the patient presents with confusion, irritability, drowsiness, and hemiparesis on same side of the injury. Initially pupillary constriction and later pupillary dilatation occurs on the same side, finally becomes totally unconscious. Death can occur if immediate surgical intervention is not done. *Features of raised intracranial pressure* like high blood pressure, bradycardia, vomiting is also seen. Occasionally *convulsions* may be present. *Wound and haematoma in the temporal region of scalp* may be seen.

CT scan-head is diagnostic. Extradural haematoma shows *biconvex* lesion (**Fig. 28.4**).

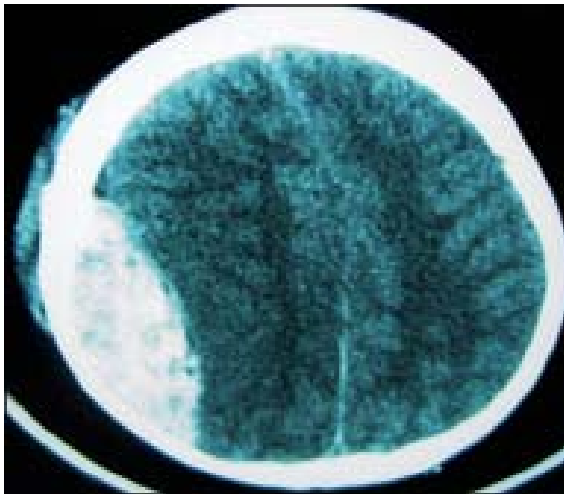


Fig. 28.4: Extradural haematoma. Note the biconvex configuration of the haematoma.

Subdural Haematoma

Types

Acute Subdural Haematoma

It is a collection of blood between the brain and dura. It is due to injury to the cortical veins and often due

to laceration of cortex of brain which bleeds and blood gets collected in the subdural space forming a haematoma. Here haematoma is extensive and diffuse. There is no lucid interval. There is a severe primary brain damage. Haematoma may be coup and contre coup type. Loss of consciousness occurs immediately after trauma and is progressive. Convulsion is common. Features of raised intracranial pressure are obviously seen—high BP, bradycardia, vomiting, etc. Focal neurological deficits or hemiparesis can occur. CT scan shows *concavoconvex* lesion.

Chronic Subdural Haematoma

It is due to the rupture of veins between dura and brain (cerebral hemispheres), causing gradual collection of blood in subdural space. It is commonly seen in elderly people following any minor trauma like fall, slipping, etc. (which might have gone unnoticed). In elderly people, brain atrophies and even minor injuries can cause shearing and bleeding from these veins. Blood collects gradually over 2-6 weeks. Plasma and cellular components get separated. Eventually cellular part gets absorbed leaving only fluid component. It is called as **subdural hygroma**. Usual haematoma collection is 60-120 ml. Often in 50% of cases it is bilateral.

Clinical Features

Common in old age, with H/O minor trauma. Patient presents with confusion, disorientation, gradually with altered level of consciousness and drowsiness. Later convulsions, features of intracranial hypertension, features of coning develops. Extensor plantar response and pupillary changes develop eventually.

Differential Diagnosis

Electrolyte imbalance, intracranial space occupying lesion.

CT scan shows *concavoconvex* lesion (**Fig. 28.5**).

Depressed Skull Fracture

It is a common neurosurgical problem among the head injuries. It means fracture depression is more than the depth of the inner table of skull (**Fig. 28.6**).

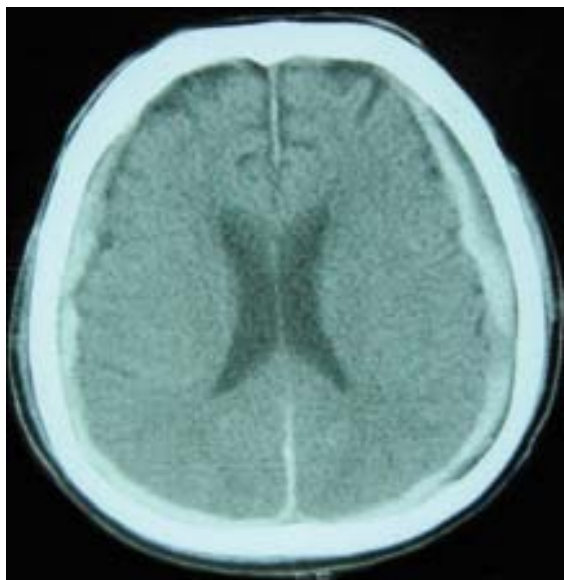


Fig. 28.5: Subdural haematoma. Note the concavo-convex configuration of the lesion.

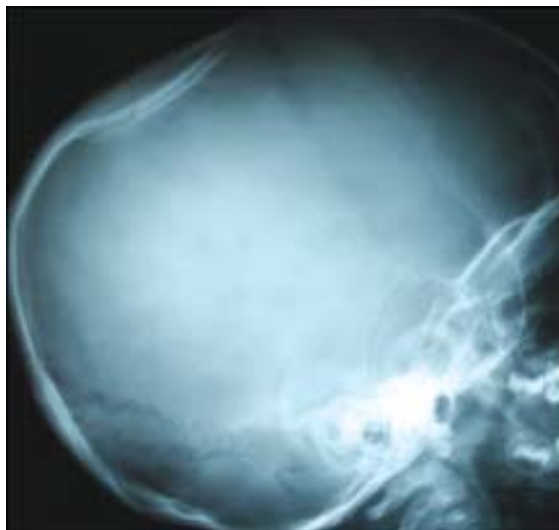


Fig. 28.6: Depressed skull fracture.

Problems in Depressed Fracture

Tear in the dura beneath; haematoma in the deeper plane; injury to the cerebrum; injury to the venous sinuses— may cause life threatening haemorrhage;

fracture should not be elevated in such occasion as it itself can precipitate bleeding; convulsions; meningitis.

EXAMINATION IN CHEST INJURIES

History

Detailed history of trauma should be asked. History of breathlessness, chest pain, haemoptysis/cough with blood (lung trauma), air way block should be asked. Pain in the ribs may be due to rib fracture. Excruciating pain on deep breathing suggests rib fracture.

Examination

General Examination

Pulse, blood pressure, cyanosis, tachypnoea, features of shock should be checked. Abdomen, limbs, head and neurological systems also be examined.

Inspection

Chest, abdomen and neck should be exposed for proper inspection. Ecchymoses, bruises in the skin

over chest wall should be inspected. Wound may often look small superficially but may be deep penetrating into the thoracic cavity. Air may be bubbling through the wound with noise. Blood, clot may be present in such wounds. Such patient often may need emergency resuscitation to maintain adequate ventilation.

Type of breathing – abdominal or thoracic and its character should be checked. Hyperpnoea, dyspnoea, altered breathing should be observed. Collapse of chest wall during inspiration and distension of part of chest wall during expiration suggests *flail chest* with paradoxical breathing, mediastinal flutter and pendular movement of the air. Tension pneumothorax causes sudden distress in breathing and sudden respiratory arrest. Emergency chest tubing or needle drainage of air from the thoracic cavity is needed.

Localised swelling due to haematoma may be evident. Diffuse puffy look suggests surgical emphysema. It may be in the chest wall, abdomen, neck both sides.

Palpation

Rib tenderness, bone irregularity, crepitus should be checked. It suggests fracture rib. With the patient standing and keeping his hands over the head, examiner keeps his one hand over the sternum and other on the spine; applying compression over thoracic cage anteroposteriorly causes pain at the site of fracture—*Compression test*.

Sternum should be examined for fracture. When such fracture is displaced upper fragment overrides lower. Spine should be examined for fracture as fracture sternum is commonly associated with fracture spine.

Flat of the hand is placed over the chest wall whenever there is diffuse swelling to feel for crepitus under the fingers. It suggests *surgical emphysema*. If it appears at the rib fracture site first, it is due to rib fracture. If it is rapidly spreading over neck, chest, abdomen, then it is due to injury to bronchus or oesophagus – *mediastinal emphysema* (**Figs 28.7A to C**).

Features of pneumothorax/haemothorax should be checked.

Percussion

Resonant on percussion in case of pneumothorax; dull in haemothorax is observed. Liver dullness is obliterated in right sided pneumothorax. Cardiac dullness is obliterated in left sided pneumothorax. Cardiac dullness is widened in haemopericardium.

Auscultation

Breath sounds is reduced in pneumothorax or haemothorax. Vocal resonance is also reduced. Heart sounds are not heard in haemopericardium.

Investigations

Chest X-ray to see fracture ribs, pneumothorax, haemothorax, haemopneumothorax.

Plural tap is often needed.

US of chest wall to confirm fluid/blood/air.



Figs 28.7A to C: Patient with puffiness of face and neck with surgical emphysema. X-ray showing surgical emphysema.

Arterial blood gas analysis (ABG) is needed to diagnose respiratory failure.

Chest Injuries

Types

(1) Crush injuries involving lung, pleura, and ribs. (2) Single rib fracture. (3) Two or more rib fractures. (4) *Steering wheel injury* - causes multiple rib fractures bilaterally often with flail chest, with fracture dislocation of upper end of sternum. (5) Stove in chest or flail chest. (6) Traumatic pneumothorax. (7) Haemothorax, haemopneumothorax, with fracture ribs. (8) Tension pneumothorax. (9) Pericardial and cardiac injuries and rupture of bronchus. (10) Associated injuries in liver, spleen, diaphragm, major vessels.

Causes

Road traffic accidents, industrial accidents, blast injuries, crush injuries, stab injuries. In children, ribs are malleable and so fracture ribs are rare. In elderly as the ribs become rigid fracture is common. First and second ribs are protected by clavicle and so fracture is uncommon. 11th and 12th ribs are floating ribs and so fracture is rare.

Factors and Pathophysiology of Chest Injuries

Hypoxia, hypercarbia, acidosis, hypovolaemic shock, pulmonary contusion syndromes, tracheo-bronchial injuries, bilateral chest injuries, chest wall injuries, diaphragmatic injuries, cardiac injuries – haemopericardium, ARDS.

Clinical Features of Thoracic Injuries

H/O trauma, painful breathing, cough, haemoptysis, pain in the chest wall, sometimes external wound may be present (in communicating wounds). Features of shock, may be seen, i.e. tachycardia, hypotension, cold

periphery. May present with respiratory distress - tachypnoea, cyanosis, respiratory difficulties. Tenderness over the fracture site may be elicited. Dullness on percussion with decreased breath sounds signifies haemothorax. Resonant with decreased breath sounds confirms pneumothorax. Surgical emphysema with palpable crepitus may be there.

Indications for Thoracotomy

Haemothorax more than 1500 ml found when ICT is placed or hourly collection in ICT of 200 ml; ICT placed showing persistent drainage of blood; Diaphragmatic injury; When associated with liver and spleen injuries; Bronchus and major vessel injury; Haemopericardium, oesophageal and thoracic duct injuries.

Complications

Infections—empyema, lung abscess, pneumonia, septicaemia; Respiratory failure; Traumatic asphyxia; Traumatic shock lung; Disseminated intravascular coagulation (DIC). ARDS (Adult/Acute Respiratory Distress Syndrome).

Flail Chest and Stove in Chest

It is fracture of *two or more consecutive ribs* with each rib having *two or more* fracture sites. Such segment is called as **flail segment**.

Stove in chest is depression of a portion of chest wall due to severe chest injury, otherwise features and management are like flail chest.

Pathophysiology

Flail segment moves separately, when compared with adjacent thoracic cage. During inspiration flail segment moves inwards (unlike normal thoracic cage which moves outward), and during expiration segment moves outwards (unlike normal cage which moves inward)

Flail chest		
Derangements	Types	Problems
Paradoxical respiration Mediastinal flutter Pendular movement of air Pulmonary contusion	Anterior—near costochondral junction Lateral—in rib shafts Posterior—safer	Cardiac arrest Respiratory failure Haemothorax Infection ARDS

causing pathophysiological derangements. This *paradoxical respiration* causes reduction in ventilatory lung surface and so respiratory dysfunction. *Mediastinal flutter*: Movement of mediastinum during different phases of respiration, often cause kinking of great vessels and sudden cardiac arrest. *Pendular movement of air* from one lung to other occurs thus preventing atmospheric air to get in to both injured and other side normal lung leading to respiratory failure. All these derangements gets aggravated by *haemothorax*, *pneumothorax* and other associated injuries.

Pneumothorax

It is presence of air between the layers of the pleura.

Classification

Pneumothorax, hydropneumothorax, pyopneumothorax, haemopneumothorax, artificial pneumothorax, tension pneumothorax.

Causes

Traumatic. Spontaneous—(1) Tuberculous. (2) Non-tuberculous: (a) Emphysematous bullae. (b) Solitary lung cyst. (c) Honeycomb or cystic lung. (d) Idiopathic.

Spontaneous pneumothorax can be acute, chronic, recurrent.

Clinical Features

Hyper resonant, absence of breath sounds, tracheal deviation.

Chest X-ray Reveals

Radiolucency on the affected side; absence of lung markings; collapsed lung margin (**Fig. 28.8**).

Tension Pneumothorax

During inspiration, air is pumped into the pleural cavity through a valvular opening in the visceral pleura and underlying injured lung. Lung collapses first and as air continuously collects in the pleural cavity, mediastinum shifts towards opposite side, decreasing the volume of the functioning lung. Further increase in the pleural pressure, reduces the venous return, atrial filling, and ventricular filling and so cardiac output

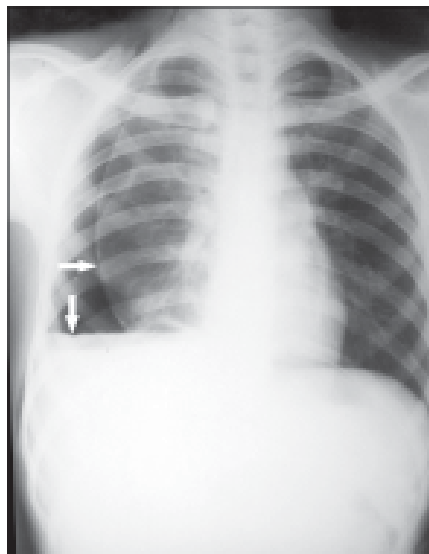


Fig. 28.8: Plain X-ray chest PA-view showing air-fluid level and collapsed lung margin (Hydropneumothorax).

and cardiac function. *It causes sudden death and hence emergency treatment is required.*

Clinical Features

Tachypnoea, decreased breath sounds, resonant on percussion with severe mediastinal shift, cyanosis.

*Once clinically diagnosed, immediately place a wide bore needle in the second intercostal space in mid clavicular line, and a sterile glove is kept on the hub (blunt) end of the needle to create a valve so as to prevent inward sucking of air from outside (**Fig. 28.9**).*

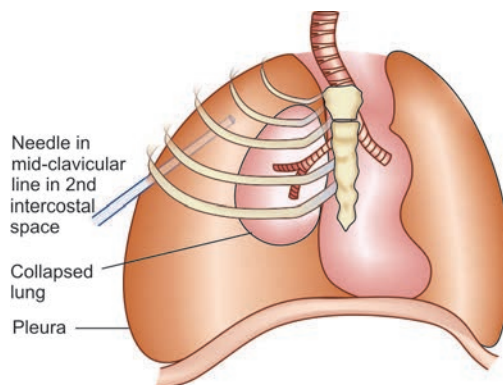


Fig. 28.9: Tension pneumothorax—Emergency decompression through 2nd intercostal space.

Haemothorax

It is blood in pleural cavity. It causes pain, shock, and also very irritant to pleural cavity. It is a good culture media for bacteria and so infection is quite common.

Causes

Trauma; Postoperative: pulmonary, cardiac, oesophageal surgeries, cervical sympathectomy; leak from CVP monitor line; Tumours of lung, mediastinum, pleura; Leaking aneurysms; Spontaneous. There may be rib fractures in traumatic haemothorax.

Clinical Features

Pain in the chest, tenderness, difficulty in breathing, dullness, diminished breath sounds; features of shock.

Complications

Infection and empyema.

Triage

Triage means 'To sort' in 'French'. Triage by committee of Trauma of the American College of surgeons.

Assessing Four Components

Physiologic response; Anatomical injury; Biomechanical injury; Comorbid factors. Triage algorithm contains Steps 1, 2, 3 and 4. Primary management consists of A, B, C, D, E and F.

Airway	Breathing
Chin lift.	100% oxygen.
Jaw thrust.	Assess bilateral chest raise.
Nasal airway.	Assess breath sounds.
Oral airway.	Use pulse oximetry.
Endotracheal intubation.	Treat flail chest, pneumothorax.
Tracheostomy.	Intercostal tube drainage
(Assess airway patency).	

Circulation Monitor vitals. Heart sounds. ECG. IV fluids Blood transfusion. Control of external bleed. Use two IV lines-14G/16G.	Disability Evaluation Neurological examination. Glasgow coma scale. Pupillary reaction. Treatment of shock.
Expose the Patient Fully Undress the patient. Hypothermia assessment. Assess injuries. Examine joints, bones, abdomen, other systems. Look for identification marks.	Fingers and Tubes Examine all orifices like P/R, P/V etc. Use required tubes like catheter, Ryle's tube.

EXAMINATION IN ABDOMINAL INJURIES

Abdominal injuries are of manifolds. It may be closed injury or open injury. It may be blunt, stab or abdominal wall injury. Open injury is obvious on examination. Closed injuries may be missed some times unless abdomen is carefully examined. Pelvic injuries often can cause life threatening torrential bleed which may be difficult to manage.

High velocity injury, compression injury, fracture spine, pelvis, penetrating injuries, gun shot injuries are different types which often can cause extensive damage.

Seat belt injuries are common in western countries. Seat belt compresses the visceral structure like bowel and mesentery causing injury. It is usually a closed/blunt injury abdomen.

History of haematuria, distension of abdomen, severe pain abdomen, bruising over the abdomen, inability to pass urine, difficulty in breathing are all important to be considered in abdominal injuries.

Often in blunt injury initially patient feels comfortable as no external wound is detected but after sometime he develops distension, pain abdomen suggesting internal organ or hollow viscus injury.

Associated chest/head/limb injuries should also be assessed by history.

Referred pain to left shoulder suggests splenic injury due to irritation of the left phrenic nerve by clot under left sided diaphragm—*Kehr's sign*.

General Examination

Features of shock, restlessness, respiration, temperature, blood pressure should be checked. Tachycardia, tachypnoea, hypotension, pallor are features of haemorrhagic shock .

Inspection of Abdomen

Patient is asked to point where exactly is the pain (*pointing sign*) and that area is inspected first. Abrasions, lacerations, bruising should be checked. Localised abdominal wall haematoma may be evident. Bruising in the abdominal wall/imprint of cloth or seatbelt suggests crushing injury of bowel against vertebral column—*London's sign* (Peter S London - Birmingham). Parallel lines (two) drawn to the line of mesentery in the abdomen divides it into upper, middle and lower parts of small bowel injury – *Monks localisation* method where bruising in these zones indicates respective (proximal, middle, distal) part of the small bowel injury (**Figs 28.10 and 28.11**).

Abdominal distension whether localised or generalised should be observed which indicates generalised or localised haemoperitoneum.

Movements with respiration may be reduced in haemoperitoneum or peritonitis.

Protrusion of umbilicus suggests dissension of abdomen by blood, pus or fluid.

Stab injury should be inspected carefully for presence of omentum, blood, and intestine. Its location,



Fig. 28.10: Penetrating injury across right side abdomen, thorax and right sided neck.



Fig. 28.11: Stab injury abdomen. It should be inspected properly.



Fig. 28.12: Urethral injury - Blood in the external meatus; extravasation of urine.

direction, margin and edge, depth should be inspected using a proper light source (**Fig. 28.12**).

Perineum and urethra should be inspected for swelling, blood in the urethra suggesting perineal injury with extravasation.

Palpation

Abdomen should be palpated for tenderness, rebound tenderness, guarding, rigidity (sign of peritonitis/irritation). Swelling in the abdomen may be localised haematoma of abdominal wall or distended bladder, paralytic ileus, localised haemoperitoneum.

Fluid thrill suggests free fluid in the abdomen. Repeated examination is needed.

Percussion and Auscultation

Obliteration of liver dullness (bowel injury), shifting dullness, and dullness lower abdomen for urinary bladder should be checked. Dullness in the left flank which is not shifting suggests splenic injury – *Ballance's sign*.

Auscultation showing absence of bowel sounds indicates peritonitis.

Rectal and vaginal examination should be done when needed.

Other systems should be examined for associated injury – thorax, limbs, and head.

Investigations

Blood parameters – Haemoglobin, packed cell volume, serum electrolytes are essential.

Plain X-ray abdomen to look for gas under diaphragm, ground glass appearance, obliterated psoas shadow, localised mass lesion (haematoma) which are significant. Chest X-ray is also to be taken.

US abdomen is essential in all abdomen traumas. It gives rapid assessment of the abdomen for haemoperitoneum and organ injuries (**Fig. 28.13**).

FAST is *Focused Abdominal Sonar Trauma*: It is rapid, noninvasive, portable bedside method of investigation focusing on pericardium, splenic, hepatic and pelvic areas. Blood more than 100 ml in cavities can be

identified. It is not reliable for bowel or penetrating injuries. It often needs to be repeated.

CT scan abdomen and chest should be done whenever needed.

Four quadrant aspirations using 19 gauge needle is done and fluid is analysed for blood, amylase, proteins, bacteria. Negative finding is of no value to rule out intraabdominal injury.

Diagnostic peritoneal lavage (DPL): It is done in case of blunt injury abdomen. Through a subumbilical lavage catheter one litre of saline is infused into the peritoneal cavity. Patient is changed to different positions side to side and later fluid content is aspirated for analysis. 10 ml or more blood; RBC count more than 1,00,000/cu mm; WBC count more than 500/cu mm; amylase level in the fluid more than 175 IU/dl; presence of bile bacteria, food – are the positive criteria. It is the procedure of choice in physiologically unstable patient. It is contraindicated in pregnancy, previous laparotomy, obesity.

Diagnostic laparoscopy is a valuable investigation.

Mesenteric angiography is needed occasionally to localise the site of bleeding.

Liver Injury

Causes

It can be due to blunt injury, stab, and gun shot injury.

Types

It can be contusion, laceration, avulsion, extension into thorax, biliary tree; associated with other organ injuries (spleen, kidney, duodenum, bowel, IVC); associated with fracture ribs.

Clinical Features

Features of shock due to severe torrential bleeding (pallor, hypotension, tachycardia, sweating). Distension of abdomen with dull flank, guarding, tenderness and rigidity are other features seen. Oliguria; tachypnoea, respiratory distress and often cyanosis is seen; Rupture of right lobe is more common than left lobe leading to haemoperitoneum; Occasionally can cause localised haematoma which will go for an abscess formation. Bile leak from the injured site can lead to *biliary peritonitis*.



Fig. 28.13: Haemoperitoneum.

Complications and Sequelae of Liver Injury

Shock and haemorrhage; liver abscess or septicaemia; bile leak, biliary peritonitis, biliary fistulas; disseminated intravascular coagulation; hepatic artery aneurysm, arterio-venous and arterio-biliary fistulas; complications of massive blood transfusion; electrolyte imbalance; respiratory complications; liver failure; **late sequelae** of liver trauma is *CBD stricture* causing obstructive jaundice. It can be managed by endoscopic stenting or by open Roux–en-Y hepatico-jejunostomy.

Liver injury

CT is diagnostic tool

Liver injury is graded depending on involvement of hepatic veins, portal system, biliary system and duodenum

Often high grade liver injury also can be managed non operatively

Push (direct compression); *Pringle* (occluding portal triad at foramen Winslow with fingers temporarily); *plug* by embolisation; *pack* the liver bed; repair of vena cava or portal vein; stenting of biliary tree and hemihepatectomy—are the treatment strategies

Splenic Injury (Rupture Spleen)

Splenic injury is common in case of road traffic accidents, and other blunt injury abdomen. Most often associated with fracture of left lower ribs, haemothorax, injury of liver (left lobe commonly, occasionally both lobes), bowel, tail of pancreas, left kidney. Injury is more common and severe in *enlarged spleen*, i.e. in

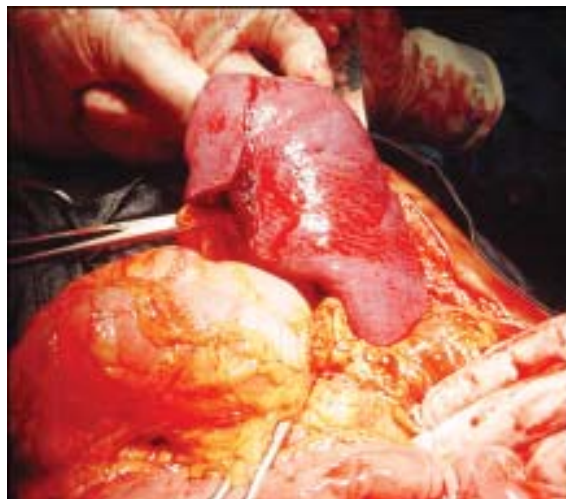


Fig. 28.14: Splenic injury.

malaria, tropical splenomegaly, infectious mononucleosis. Spontaneous rupture of spleen can occur in malaria and infectious mononucleosis (**Fig. 28.14**).

Types of Injury

1. *Splenic subcapsular haematoma*: After initial injury patient remains asymptomatic but this haematoma later ruptures after few days causing torrential haemorrhage.
2. *Clean incised wound over the surface*: This can be treated by splenorrhaphy.
3. *Lacerated wound*.
4. *Splenic hilar injury* causes torrential haemorrhage, may even cause death. So immediate surgical intervention and splenectomy is done.
5. *Splenic injury associated with other injuries*.

Presentation

Rapid development of *shock and fast deterioration* can occur. Even death can occur sometime, which is often due to splenic hilar vessel injury, where *emergency surgery and splenectomy is mandatory*. In other types, features of shock (pallor, tachycardia, restlessness, hypotension), pain, tenderness, and abdominal rigidity in left upper quadrant is seen. Later there will be abdominal distension due to haemo-peritoneum. There is dullness in the left flank which does not shift, as the collected blood gets clotted. *Dullness without shifting—Ballance's sign*. Clot collected under the left side of the diaphragm irritates it and the phrenic nerve causing *referred pain in the left shoulder—Kehr's sign*. *Seagesser's tender point* between left sternomastoid and scalenus medius. There may be left sided haemothorax with fracture of ribs. Delayed presentation is also possible due to formation of subcapsular haematoma which later gives way. Initially gets temporarily localised by greater omentum, later giving way leading to torrential bleeding. Blood clot temporarily seals off the bleeding which later gets dislodged causing severe bleeding. This time period in between is called '*latent period of Bandet*'.

Features of other abdominal organ injuries like of left lobe of liver, tail of pancreas, left kidney, left sided colon, small bowel, may be present. Diaphragm and left sided lung injury, fracture ribs may be other associations.

Plain X-ray

Plain X-ray shows—Obliteration of splenic outline and left sided psoas shadow; indentation of fundic gas shadow; fracture ribs; elevated left side diaphragm. US abdomen and CT are diagnostic.

Complications of Splenic Injury

Haemorrhagic shock; DIC; sepsis; splenic artery pseudoaneurysm; splenic arteriovenous fistula; pancreatitis.

Injuries to Kidney

Commonly it is due to a blunt injury. Often it is associated with other abdominal injuries of liver, spleen, bowel, mesentery, etc. *Per se renal injury is extraperitoneal.*

Types

Small subcapsular; Large subcapsular; Cortical laceration; Laceration with perinephric haematoma; Medullary laceration with bleeding into the renal pelvis; Corticomedullary complete rupture; Hilar injury (most dangerous).

Clinical Features

Features of *shock*; *Haematuria*—may be mild to profuse depending on the type of injury; *Clot colic*; Bruising, swelling and tenderness in the loin; Paralytic ileus with abdominal distension occurs due to retroperitoneal haematoma implicating splanchnic nerves.

Investigations

IVU (high dose) is the investigation of choice. Function of not only the injured kidney but also of the contralateral kidney can be seen assessed. Because it is observed that most often opposite renal artery undergoes reflex spasm, temporarily ceasing the function of the contralateral kidney. **US abdomen:** Done to see the type of injury, amount of haematoma and other associated injuries in the abdomen. US should be repeated at regular intervals to see the progress (at 12-24 hourly). Blood urea and serum creatinine should be repeated again at regular intervals. Blood grouping and cross matching for blood transfusion.

Complications

Clot retention in the bladder and may go for renal failure; Pararenal pseudohydronephrosis; Infection; Perinephric abscess; Aneurysm of the renal artery; Renal failure.

Pancreatic Trauma

It is rare because of its anatomical location—retroperitoneum. Its injury is usually associated with injuries to liver / duodenum / spleen / portal system / biliary system / kidney. Deep force in epigastrium may cause crushing of body of pancreas against vertebra—closed injury. Penetrating injury may cause direct sharp injury of pancreas.

Types

Parenchymal contusion/laceration without duct disruption; Parenchymal injury with duct disruption; Complete transection of pancreas; Massive destruction of pancreatic head.

Classification

Pancreatic injury

Class I Capsular damage; minor parenchymal injury
Class II Transection of duct in body or tail – partial or complete
Class III Major duct injury in pancreatic head or intrapancreatic CBD injury

Duodenal injury

Class I Contusion, hematoma, partial thickness injury
Class II Full thickness duodenal injury
Class III Full thickness duodenal injury with
 1. More than 75% circumference injury
 2. Full thickness duodenal injury with extrapancreatic CBD injury

Combined pancreaticoduodenal injury

Type I Class I injury to both organs/class I injury of one organ with class II injury to other
Type II Class II injuries of both organs
Type III Class III injury to one organ with less severe injury to other
Type IV Class III injury to both organs

Features

Pain in epigastrium; Features of associated injuries; Features of shock; Rise in serum amylase level is common.

Investigations

CT scan is diagnostic; ERCP to confirm duct disruption; Assessment of blood loss and other injuries.

Complications

Haemorrhage; septicaemia; pancreatitis – acute/recurrent; pseudocyst formation; pancreatic fistula; pancreatic abscess.

Rupture Bladder

Causes

Blow, kick or fall; road traffic accidents; stabs, gunshot injuries; endoscopic trauma; diathermy; instrumentations.

Types

Type I: Intraperitoneal rupture—20% common. It occurs in fully distended bladder due to blow, kick, or fall (**Fig. 28.15A**).

Type II: Extraperitoneal rupture: 80% common. It is due to road traffic accidents, golf playing, fall over the manhole. Its features and management are same as rupture of posterior (membranous) urethra (**Fig. 28.15B**).

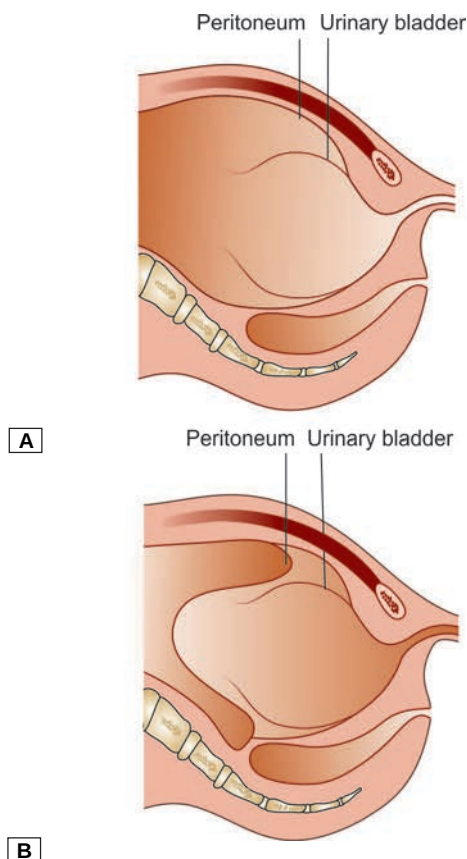
Intraperitoneal Rupture

It is 20% common. *It occurs in full bladder.*

Clinical features: Sudden pain in suprapubic region. Shock and syncope; diffuse abdominal pain. Urine leaks into the abdominal cavity causing distension of abdomen. Later it causes features of peritonitis, with guarding, rigidity, tenderness and rebound tenderness, dull flank. *Patient does not have the desire to micturate.*

Investigations: (1) Plain X-ray shows ground glass appearance. (2) Peritoneal tap to collect urine. (3) After passing a small catheter gently per urethra, water soluble iodine dye is passed to visualise the tear in the bladder and entry of the dye into the free peritoneal cavity. This can be done now through 'C-ARM image' intensifier easily. (4) US abdomen to look for other injuries in the abdomen.

Complications of rupture bladder: Cystitis and pyelonephritis; peritonitis; pelvic abscess; fistula



Figs 28.15A and B: (A) Intraperitoneal rupture. (B) Extraperitoneal rupture.

formation (vesicovaginal or rectovesical); paralytic ileus; haemorrhage. *Without surgery mortality is 100%.*

Extraperitoneal Rupture of the Bladder

It is 80% common. It occurs in road traffic accidents, in a non distended bladder.

Clinical features: There is collection of urine and blood in the extraperitoneal space in front, with fullness, diffuse pain and tenderness in lower abdomen. Swelling is seen in the scrotum or labia, and abdominal wall. Strangury, inability to pass urine and often blood in the external meatus is noted. Features of shock and other associated injuries may be noted.

Investigations: Plain X-ray pelvis shows fracture. Cystogram shows leak from the bladder.

During cystoscopy—(1) Bladder cannot be distended. (2) Endoscopy light may be shining through the abdominal wall. (3) Irrigating fluid cannot be retrieved back. Other associated urethral injury is looked for.

Bladder injury can also occur during hysterectomy, (both abdominal and vaginal), surgery of colon or rectum, repair of direct inguinal or femoral hernias.

Urethral Injury

Classification

Depending on site of rupture (Fig. 28.16): (1) Rupture of the membranous urethra. (2) Rupture of the bulbous urethra.

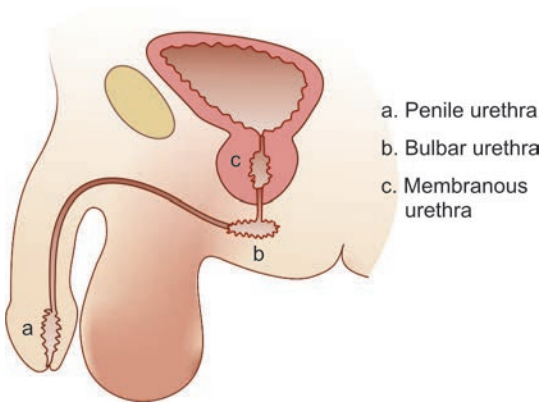


Fig. 28.16: Common sites of urethral injuries. Membranous and bulbar urethra are common sites.

Depending on circumference of the urethral wall involved: (1) Complete. (2) Incomplete.

Depending on the thickness of the urethra involved: (1) Total. (2) Partial.

Rupture of Membranous Urethra (Prostatic Urethra/Posterior Urethra)

Causes: It is usually associated with pelvic fracture, commonly due to road traffic accidents. Injury can also occur during instrumentation, calculus passage and catheterisation; In prolonged labour, due to long standing pressure on the urethra by foetal head.

Prostate is attached to pubis by puboprostatic ligament and disruption of puboprostatic ligament with

complete rupture of urethra can lead to **floating prostate**. Injury can lead to incomplete rupture of urethra or may be associated with extraperitoneal rupture of bladder.

Based on ascending urethrogram posterior urethral injury is classified as (*Macculum-cola-pinto classification*)—Type I: Elongation of posterior urethra, but intact. Type II : Prostate “plucked off” membranous urethra with extravasation of urine above sphincter only **“Floating prostate”**. Type III: Total disruption of urethra with extravasation both above and below the sphincter.

Clinical features: (1) Blood in external meatus. Failure or difficulty in passing urine. (2) Extravasation of urine to scrotum, perineum and abdominal wall. (3) **Shock** with pallor, tachycardia, hypotension. (4) Features of associated injuries like head injury, thorax and or abdominal organs which take priority in initial phases of management. (5) On P/R examination, prostate may be felt high or may not be palpable at all. It signifies **floating prostate**.

Investigations: X-ray pelvis to see for fracture. US abdomen to see pelvis and other injuries. **Urethrogram** is done to see the site and type of tear (often reserved to do at later stage) (**Fig. 28.17**).

Complications: Urinary incontinence; impotence; stricture urethra; infection.



Fig. 28.17: Pubic bone fracture with SPC – Malecot catheter.

Rupture of Bulbous Urethra (Anterior Urethra)

Usually due to a fall astride a projecting object, like in sailing ships, cycling, over loose manhole cover, gymnasium.

Clinical features: Triad—(1) Blood in external meatus (Urethral haemorrhage). (2) Perineal haematoma. (3) Retention of urine. Rupture may be *complete or incomplete/Total or partial*.

Investigations: X-ray pelvis, and US abdomen. Condition is diagnosed clinically.

Complications: Infection; extravasation of urine; stricture urethra.

Abdominal Compartment Syndrome

There is sudden increase in intraabdominal pressure which causes decreased venous return to heart (pressure more than 85 cm of water). It leads into increased peak inspiratory pressure, hypoxia, hypercarbia, decreased urine output, hypotension due to decreased venous return to heart.

Intra-abdominal pressure grading (Burch) in cms of water

I—10-15 cm of H₂O

II—15-25

III—25-35

IV—more than 35

Abdominal compartment syndrome (ACS)		
Causes	Features	Management
Postoperative ileus Acute abdomen Acute gastric dilatation Laparoscopic procedures Intestinal obstruction	Hypoxia, hypercarbia Decreased urine output Hypotension Tense abdomen-distended Decreased venous return Bowel ischaemia Cardiac arrest	Bladder pressure assess Ryle's tube aspiration Resuscitation ICU care Surgical decompression

Examination in Intracranial Diseases

History

Medulloblastoma, glioblastoma affects younger individuals. Acoustic neuroma occurs after 30 years.

History of trauma earlier may cause late subdural haematoma or intracranial abscess causing convulsions, localising features.

Frontal sinusitis, otitis media can cause intracranial abscess later. Such history is reliable.

History of convulsions, nature of its occurrence in detail should be asked.

History of loss of sensation, weakness in the limb suggests neurological deficit. It suggests cerebral pathology on opposite side.

In co-ordination on the affected side (walking) suggests cerebellar disease.

Change in the personality, retarded features, loss of memory and concentration are features of frontal lobe syndrome or tumour.

Headache (initially early morning, later generalised) is the common feature in intracranial tumours. It is unilateral on the side of the lesion. Posterior lesions show occipital headache which radiates to neck. Pituitary tumours cause bitemporal headache.

Early morning vomiting is usual without any nausea, and is aggravated by straining.

Change in the vision may occur in many tumours as part of papilloedema, pituitary and, temporal tumours.

Drowsiness, neck stiffness, severe headache, vomiting, are the features of possible coning.

General Examination

Gigantism, acromegaly, Cushing's syndrome are features of pituitary tumour (**Figs 29.1A and B**). Bradycardia, hypertension are features of raised intracranial pressure.



Figs 29.1A and B: Acromegaly features due to pituitary tumour.

Nervous System Examination

Detailed nervous system examination is needed.

Mental status, speech, coordination, orientation, vision should be checked in detail.

Cranial nerves should be checked properly.

Examination of Cranial Nerves

Cranial nerve palsies that commonly presents in head and face is being discussed in this chapter.

Cranial nerves are – Olfactory; Optic; Oculomotor; Trochlear; Trigeminal; Abducent; Facial; Auditory; Glossopharyngeal; Vagus; Accessory; Hypoglossal nerves (Mnemonic—On Old Olympus Towering Tops A Finn And German Picked Some Hops).

Olfactory: Sense of smell is tested with cloves, peppermint etc. Meningioma of olfactory groove or of base of frontal lobe causes *anosmia*; *parosmia* or perversion of sense of smell is due to uncinate process lesion.

Optic: *Visual acuity* (ability to read), *visual fields* (peripheral vision to be checked in one eye and compared to examiner's), *Colour vision* using charts. Blindness in one half of visual field is called as *hemianopia*. If it is seen in same half of each visual field, it is called as homonymous hemianopia (optic tract lesions and radiotherapy effects). In optic chiasmal lesion bitemporal hemianopia (outer half of each field is affected) is seen. It may be due to pituitary tumour or suprasellar cyst. Pituitary tumour presses chiasma from below causing upper quadrantic hemianopia; whereas suprasellar cyst pressing from above causes lower quadrantic hemianopia.

Oculomotor: It supplies all extrinsic muscles of eyeball except superior oblique (trochlear), lateral rectus (abducent), levator palpebrae superioris and muscle of accommodation. In oculomotor nerve palsy eye looks downwards and outwards with ptosis (drooping of upper eyelid) and fixed pupil. Superior rectus - to look up; medial rectus - to converge; inferior rectus - to look down; inferior oblique - to look up and out. Complete paralysis causes - ptosis (paralysis of levator palpebrae superioris); externoinferior squint (due to unopposed action of external rectus and inferior oblique); inability to move eyeball inwards and

outwards; dilatation pupil; loss of light and accommodation reflexes; diplopia.

Trochlear: It supplies superior oblique muscle. When it gets damaged turning eye downwards and outwards is defective and patient looks inwards with diplopia below the horizontal line.

Trigeminal: Sensory supply is to entire one side of the face by three divisions - ophthalmic - upper; maxillary - middle; mandibular - lower. Ophthalmic division also supplies conjunctiva. Maxillary branch supplies mucous membrane of nose, pharynx, roof of mouth, soft palate and tonsil; mandibular division to tongue, lower teeth, mucous membrane of the mandible. Sensations should be checked in this place. Conjunctival reflex, palatal reflex will be altered. In trigeminal neuralgia there is hyperaesthesia with touch becoming pain. During the period of neuralgic attack entire area is hyperaesthetic. *Only certain trigger zones of Patrick* is hyperaesthetic in between attacks. Motor supply to masseter, pterygoids and temporalis is from mandibular branch. Clenching the teeth will confirm the same. While opening the mouth widely jaw deviates towards the affected side due to weakness of pterygoids. Taste from anterior 2/3rd is through lingual nerve via chorda tympani from geniculate ganglion. Sweet (sugar), sour (acid), salt (salt) and bitter (quinine) tastes are checked. Salt and sweet in the tip of the tongue (through chorda tympani); sour is in lateral margin of tongue through trigeminal nerve; bitter is in posterior tongue through glossopharyngeal nerve.

Abducent nerve supplies the lateral rectus muscle of the eye. Turning of eye outwards is defective in its paralysis and attempt to look side will cause diplopia.

Facial: It supplies muscles of facial expression. It is motor nerve. Supranuclear palsy causes lower facial palsy; infranuclear palsy causes entire facial nerve palsy. Features includes—Eyelids cannot be closed; whistling is defective; angle of the mouth deviates; wasting of the muscles of that side; wrinkling of eye is defective; inability to close the eyes properly.

Auditory: It supplies cochlea and semicircular canals. *Weber's tuning fork test* is used to rule out conductive deafness. After placing the tuning fork on the forehead louder sound is heard on the side of conductive

deafness. Tuning fork is placed on mastoid to get louder sound in conductive deafness in *Rinne's test*. In sensory deafness there is no change in sound appreciation. Assessing the response to changes in temperature in the external meatus—*calorie test* is used to check the sensitivity of the vestibular apparatus.

Glossopharyngeal: It is sensory to posterior third of the tongue (and also carries bitter taste checked by using quinine) and to mucous membrane of pharynx. It is motor to middle constrictor. *Gag reflex* can be elicited by stroking the back of oropharynx.

Vagus: It is motor to soft palate, pharynx and larynx and sensory to gut, heart and lungs. After opening the mouth patient is asked to say 'Aahh'. Soft palate arches upwards symmetrically. In paralysis of one side, it will not arch symmetrically and uvula gets pulled towards functioning (*opposite*) side. Change in voice, inability to cough and vocal cord palsy in indirect laryngoscopy are the other features.

Spinal accessory: Wasting of sternomastoid and trapezius is obvious. When chin is pushed towards opposite side against resistance weakness can be appreciated; shrugging of the shoulder against resistance is defective when checked from behind.

Hypoglossal nerve: It is motor to tongue. When it is paralysed, wasting of tongue is seen on the same side; tongue deviates towards *same side* while protruding out.

Nystagmus

It is involuntary oscillations of eyeball. It is due to cerebellar lesions or vestibular lesions. Eyes persistently turning towards one side is called as conjugate deviation. In cerebral pathology, eyes are directed towards paralysed side and away from the irritating side. In pontine lesions eyes are directed towards irritative side, away from paralytic side. Skew deviation of eye is one eye looking upwards and other downwards – is seen in some cerebellar pathology.

Cerebellar Lesion

It is checked by many methods – 1) Extended arm with forefinger is brought to tip of the nose with patient closing his eyes. 2) Walking in a straight line. 3) Rapid pronation and supination with forearm at right angle

is not possible – *adiadochokinesia*. 4) With eyes closed patient is asked to stand with feet very close together; patient sways towards the side of the lesion – *Romberg sign*. 5) Nystagmus.

Muscle tone, rigidity, flaccidity should be checked. Power of muscle should be graded. Wasting of muscle should be observed

Neck rigidity is an important sign (*Kernig's sign*) to be confirmed. Tics, athetosis, tremors of hands should be checked.

All skin sensations; joint position sense; size, shape, form of objects given (*stereognosis*) should be checked.

Reflexes

Ankle jerk (S_1, S_2) is checked by gently stroking the Achilles tendon with foot dorsiflexed which causes sudden contraction of calf with rapid plantar flexion of the foot.

Knee jerk ($L_{2,3,4}$) is checked by a blow on patellar ligamentum of the knee which is kept over opposite knee or held by the examiner will cause brisk contraction of the quadriceps. Knee/ankle jerks are exaggerated in pyramidal tract lesion.

Triceps jerk (C_7) is checked by tapping above the olecranon with the elbow flexed causing contraction of triceps.

Biceps jerk ($C_{5,6}$) is checked by holding patients elbow with left hand and placing thumb over biceps tendon and tapping over the thumb will elicit the jerk.

Cremasteric reflex (T_{12}) is done by scratching the skin over upper inner part of thigh to draw testis upwards. It is absent in pyramidal tract lesion.

Abdominal reflexes ($T_{7,11}$) are elicited by strokes over the abdomen parallel to costal margins and iliac crests causing umbilical movements. It is absent in pyramidal tract lesion.

Ankle clonus ($S_{1,2}$) is checked by sudden dorsiflexion of foot with knee flexed slightly and heel off the ground causing oscillations of foot.

Patellar clonus ($L_{2,3,4}$) is checked with knee extended and pushing patella downwards by holding it between the thumb and fingers to develop continuous clonic movements of patella.

Relevant Other Examinations

Ear, nose and mouth should be examined.

Respiratory system, abdomen should be examined.

Investigations

Blood—haemoglobin, ESR, total count.

Chest X-ray.

CT scan to see the lesion, extent, size. Often MRI may be needed.

Lumbar puncture – It should not be done if there is papilloedema. Normal pressure is 120 mm of H₂O. If it is more than 160 mm of H₂O, CSF fluid collected should be very limited, otherwise coning due to herniation of temporal lobe through tentorium cerebelli or medulla through foramen magnum can occur. CSF is analysed for cells (normal cells are < 50; it is raised more than 100 in gliomas); proteins (normally it is 20–30 mg %), it is raised in meningiomas, cerebral abscess.

X-ray skull: It is replaced by CT and MRI. Earlier X-ray and angiogram were essential investigations. **Features of increased intracranial pressure** are - separation of all sutures in children; silver beaten appearance of skull; thinning of posterior clinoid fossa. Erosive, hypertrophic and sclerotic features with vascular grooves and calcification are typical of meningioma. Astrocytoma, 40% oligodendrogliomas, 50% craniopharyngiomas and tuberculomas show calcification. Widening of sella turcica with patchy calcification is seen in suprasellar cyst. Calcified pineal body shift is a feature of displacement by large tumour.

Combined carotid and vertebral angiography is useful investigation to find out vascularity, extent, etc.

Ventriculography, encephalographies were used earlier; now not used.

Biopsy using Dandy's brain cannula after doing burr hole is useful to have histological confirmation.

Radioisotope scan of brain using technetium^{99m} or I¹³¹ is useful to identify primary tumours, metastatic tumours, brain abscess, inflammatory pathologies, assessing the blood flow. It has got 80–90% sensitivity without any morbidity.

Intracranial Abscess

Types

Extradural abscess: Caused by - Osteomyelitis of skull, middle ear infection, frontal sinusitis. **Pott's puffy tumour** is infection and inflammation of the scalp. There is acute localised headache and tenderness in

the skull, localised pitting oedema of the scalp usually in the frontal region.

Subdural abscess: Is caused by septic thrombophlebitis from the frontal sinusitis or other infections. It is often very severe with extension into the venous sinuses.

Intracerebral abscess: Is caused by (1) Extension from middle ear or sinuses. (2) Blood born infection. (3) After intracranial injuries.

Common sites: Temporal lobe, cerebellum, frontal lobe. It can be: *a. Acute*—There is acute septic encephalitis without pus formation. It may cause ventriculitis or localised abscess formation. *b. Subacute*—Commences at 3 weeks, by the formation of a glial wall, i.e. thickness is more near the cortex and less towards ventricle. *c. Chronic*—Occurs in 6 weeks with thick wall which may persist and may get enlarged behaving like a space occupying lesion. *d. Metastatic*—Abscess in brain occurs either in cerebrum (parietal or temporal lobes) or in ventricles (*Ventriculitis* is more dangerous and often fatal).

Clinical Features

Evidence of focus of infections are seen, i.e. middle ear (CSOM), sinusitis. Focal neurological features are seen, depending on the location of abscess. In temporal lobe abscess there will be dysphasia, contralateral hemiparesis; in cerebellar abscess, all cerebellar symptoms are seen (**Fig. 29.2**). Epilepsy; Features of raised intracranial pressure: a) slow pulse, b) rising BP, c) headache and vomiting, d) papilloedema, e) deterioration in level of consciousness, f) visual disturbances are other features.

Differential Diagnosis

(1) Intracranial tumour, (2) Tuberculoma, (3) Meningitis.

Lumbar puncture should be avoided in acute abscess as coning can occur.

Subarachnoid Haemorrhage

It is a type of intracranial haemorrhage where bleeding occurs into the subarachnoid space usually from basal cisterns. It is usually spontaneous.

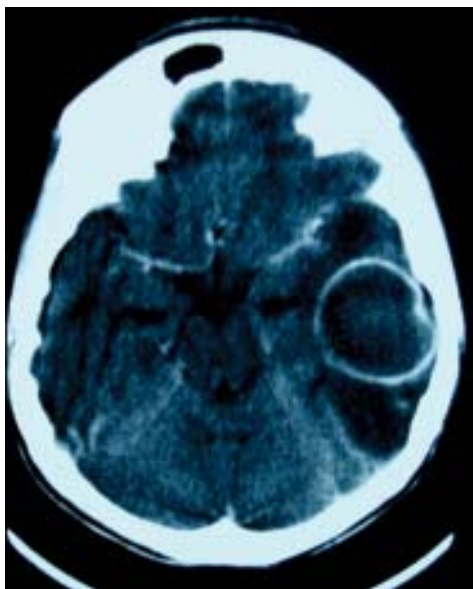


Fig. 29.2: Intracranial abscess – CT picture.

Causes

Intracranial aneurysms—commonest cause (50%). Hypertension; A-V malformations; Blood dyscrasias; Anticoagulant drugs; Brain tumours (malignant).

Clinical Features

Sudden onset of severe headache with vomiting. Features of raised intracranial pressure; Photophobia; Neck stiffness; Focal neurological deficits: Hemiplegia, dysphasia; eye changes: ptosis, dilated pupil, changes in the eyeball movements. Sudden loss of consciousness; Features of brain oedema and cerebral ischaemia.

In 40% of recovered patients, rebleeding occurs in 6-8 weeks which is commonly fatal.

Differential Diagnosis

Meningitis; coning due to any cause.

Intracranial Aneurysms

Types

1. Subclinoid type occurs in the internal carotid artery within the cavernous sinus. It causes ptosis, defective external ocular movements, and 5th nerve palsy. It

can cause carotidocavernous fistula. **2. Supraclinoid type** is common type. (1) *Berry's aneurysms*: A *congenital type* occurs in circle of Willis in relation to internal carotid artery [40% (most commonly at the origin of posterior communicating artery)], anterior communicating artery, middle cerebral artery, vertebro-basilar artery. It occurs due to weakness in the media of major arteries. (2) *Acquired aneurysms* due to atheromas, hypertension, etc. (3) *Mycotic aneurysms* occur due to infection in the wall of cerebral vessels as a result of any bacteraemia. Common sites are peripheral branches of middle cerebral artery.

Presentations

Subarachnoid haemorrhage; pressure effects; convulsions; eye and pupillary signs (**Fig. 29.3**).

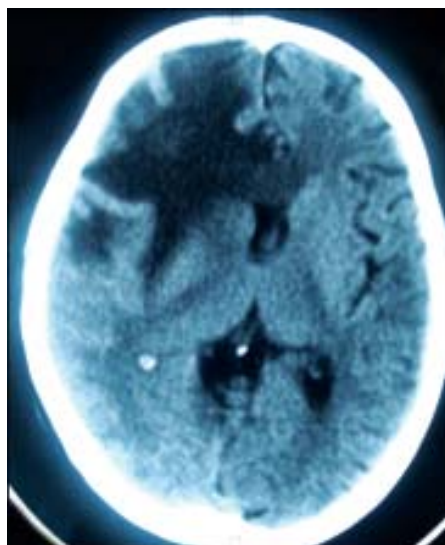


Fig. 29.3: CT scan showing brain haemorrhage due to ruptured aneurysm.

Intracranial Tumours

Secondaries are the commonest malignant tumour in the brain. Metastasis occurs usually from lung (commonest), nasopharynx or from any other organ in the body.

Primary Brain Tumours

Gliomas (43%): (a) Astrocytomas are the commonest type. They are usually malignant. They can occur

anywhere in the cerebral hemispheres, medulla, and brainstem. They can be *diffuse, solid, or cystic*. They contain star shaped cells resembling adult neuroglial cells. Astrocytic gliomas are graded as *Grade I, II, III, and IV as per the quantity of adult and primitive cells*. **(b) Oligodendrogliomas.** **(c) Spongioblastoma polare** arises from the primitive spongioblasts affects optic chiasma, third ventricle, hypothalamus, etc. They are irremovable but are radiosensitive. **(d) Medulloblastoma** occurs in children, affecting vermis of the cerebellum which grows rapidly with seedling elsewhere in the brain. **(e) Ependymomas:** Cells here resemble ependymal cells. It can occur throughout the cerebral hemispheres (**Fig. 29.4**).

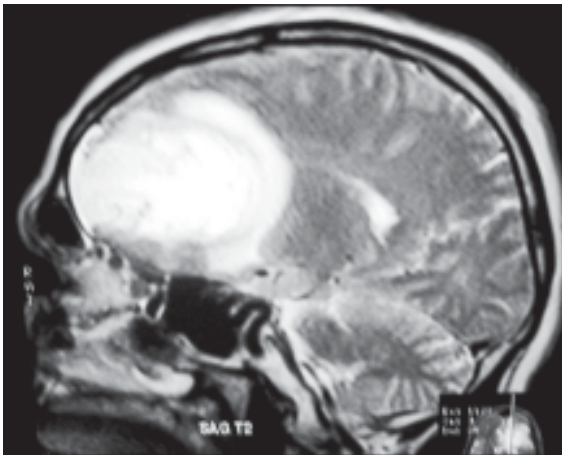


Fig. 29.4: MRI showing glioma.

Meningiomas (18%): They are usually globular, arising from the arachnoids. Tumour gets attached to the dura. It gets blood supply from dural arteries and veins, from emissary veins, veins of diploe and scalp. Tumour cells invade the bone along these veins, causing bone destruction and reactive hyperostosis. Meningiomas can be *calcified, fibroblastic, endothelial and angioblastic*.

Sites

(1) Parasagittal, (2) Frontobasal, (3) Posterior fossa, (4) Choroid plexus.

Microscopic: It contains whorls of spindle cells, with central hyaline material, with Psammoma bodies.

Schwannoma (8%): Common in auditory nerve also called as *acoustic neuroma*. It occurs in the internal auditory meatus which projects into the cerebello-pontine angle (C- P angle), compressing 5, 6, 7, 8th nerves. It presents with compressive features like unilateral deafness, trigeminal neuralgia, squint, cerebellar compression syndrome.

Pituitary tumours (12%).

Craniopharyngiomas (5%).

Blood vessel tumours (2%).

Clinical Features

Initial period of silent growth; Focal syndromes with epilepsy; Raised intracranial pressure with headache, vomiting, deterioration of level of consciousness, altered vision, slow pulse, high BP, papilloedema; Brain displacement and stage of coning.

Specific Features

Frontal lobe tumours: Personality and emotional changes, epilepsy of generalised type, contralateral facial weakness.

Parietal lobe tumours: Jacksonian epilepsy, progressive hemiparesis, astereognosis, acalculia.

Occipital lobe tumours: Aura of flashing of light in contralateral field, homonymous hemianopia.

Temporal lobe tumours: Progressive aphasia, visual, auditory, smell and taste hallucinations, hemiparesis, superior quadrantic hemianopia.

Midline tumours: Produces bilateral hydrocephalus.

Tumours of the third ventricle (colloid cyst is common): Causes bilateral hydrocephalus, progressive cerebral atrophy, dementia, sexual precocity, endocrine disturbances.

Pineal tumours: Causes precocious puberty.

Cerebellar vermis tumours: Usually medulloblastomas, occur in young children, presents with progressive hydrocephalus and features of herniation of cerebellar tonsils through foramen magnum.

Cerebellar hemisphere tumours: Commonly are astrocytomas, produce cerebellar syndromes, nystagmus, etc. (**Fig. 29.5**).

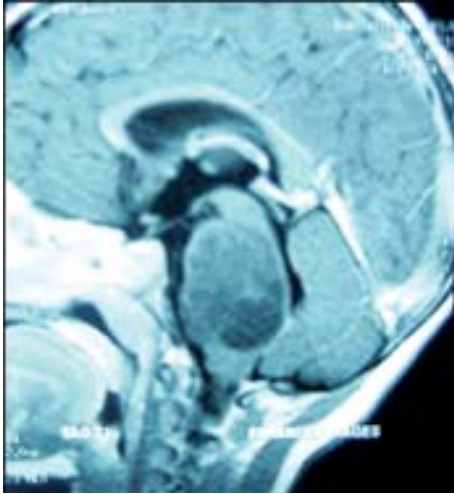


Fig. 29.5: MRI showing pontine tumour.

Pituitary Tumours

Classification I

Eosinophil (Acidophil) adenomas: Tumour is usually small. Rarely it causes compressive features. It secretes excess growth hormone causing acromegaly in adults and gigantism in children.

Chromophobe adenomas: Are common in females and in the age group (20-50 years). Initially it is *intracellular* and after sometime becomes *suprasellar*. Later it extends *intracranially* often massively, causing features of intracranial space occupying lesion. It presents with myxoedema, amenorrhoea, infertility, headache, visual disturbances, bitemporal hemianopia, blindness, intracranial hypertension, epilepsy. **Differential diagnosis:** Meningiomas, aneurysms. CT scan, Angiogram, X-ray skull are diagnostic.

Basophil adenomas: Are usually small. They secrete ACTH and presents as Cushing's disease with all its features.

Prolactin secreting adenomas: Causes infertility, amenorrhoea and galactorrhoea.

Classification II

1. Hypersecreting
2. Hyposecreting by compression and atrophy.

Classification III

1. Micronodular: Tumour size less than 10 mm.
2. Macronodular: Tumour size more than 10 mm.

Stages

1. Stage of intrasellar development.
2. Stage of suprasellar extension.
3. Stage of massive intracranial extension.

Craniopharyngiomas

They are large masses with cystic cavities, lined by ciliated epithelium containing cholesterol crystals. Areas of calcifications may be present and coral-like masses may be formed. They are adherent to the basal arteries and adjacent nerves. They are *irremovable*. They are tumours of sellar region.

Clinical Features

Intrasellar craniopharyngiomas inhibits sexual maturation causing obese, impotent dwarf with bitemporal hemianopia (due to compression of optic chiasma)—**Frolich's syndrome**. *Suprasellar craniopharyngiomas* produces Frolich's syndrome; pressure on hypothalamus which controls sleep and water metabolism (causes somnolence and diabetes insipidus). *Massive intracranial extension* causes intracranial hypertension and also hydrocephalus by obstructing CSF flow (**Fig. 29.6**).

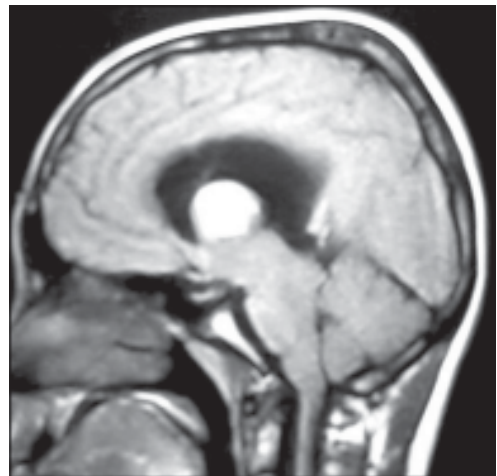


Fig. 29.6: CT scan showing craniopharyngioma.

Examination of Bone and Joint Injuries and Pathology

EXAMINATION OF BONE INJURIES

History

Age: Greenstick fracture commonly occurs in children; dislocations in adults.

Types of trauma: (1) Direct by tapping, crushing. (2) Indirect by twisting, bending, with compression force. (3) Muscular due to undue contraction of muscle against resistance—patellar fracture.

Pain: It is present at the site of fracture may be severe. It is much more unbearable in dislocation of the joint.

Loss of function of the part either lifting or walking depends on the site of the fracture.

Swelling at the site of fracture is common due to soft tissue contusion.

Deformity is common depends on the type of fracture.

Examination

Inspection

Swelling is due to soft tissue injury or haematoma. Its site, size, extent should be noted. Oedema at fracture site is common. Displaced fracture segment may be felt as bony swelling or projection.

Skin overlying swelling or fracture site: There may be ecchymoses, bruising, discolouration, discharge, and blood in the wound. Fracture may communicate outside with skin and soft tissue disruption—called as *compound/open fracture*. Fracture which is not communicating is called as *simple closed fracture*. Wound should be explored with all aseptic precautions.

Deformity is specific for specific fractures.

Attitude of the limb: There are specific attitudes for specific fractures. Externally rotated limb is seen in fracture neck of femur.

Shortening: It is due to overlapping of fracture segments.

Palpation

Local bony tenderness is elicited by palpating the entire length of the bone and comparing. Bony irregularity is checked by elevation/gap. It is a definitive sign of fracture.

Abnormal movement between fracture segments is a definitive sign but eliciting should be with gentle. It is useful in old fractures.

Crepitus is grating sensation between cut ends and is painful. Crepitus is also elicited in other conditions like surgical emphysema.

Pain may be elicited in the fracture site by rotation, squeezing/pressure/springing.

Absence of transmitted movements is typical of fracture.

Measurements both longitudinal and circumferential are checked. Sound normal limb should be kept in the same position as affected limb prior to measurement. Sound limb should be measured first. Circumference should be measured at same level from a bony point and compared.

Movements both active and passive should be checked. It is totally absent in dislocation.

Associated injuries should be looked for. Often that is more important than fracture itself like vessel, nerve injury, and major internal organ injuries.

Features of shock due to haemorrhage, pain should be checked.

Pathological fractures needs special mention as cause for fracture is something else but may be precipitated by minor trauma. Examples are—osteogenesis imperfecta (brittle bones); osteopetrosis (marble bones) in infants; osteomyelitis and solitary bone cyst in children; osteosarcoma in young; multiple myeloma, hyperparathyroidism, osteoclastoma in adults; secondaries, old age, Paget's disease in elderly.

Investigations

X-ray of site/part: Both AP and lateral view should be taken. Fracture/displacement/tilt/twist should be looked for. Oblique view in scaphoid fracture is needed (Fig. 30.1).

Blood/serum for calcium, acid phosphatase, alkaline phosphatase, tumour markers for specific diseases. Immunoglobulins are analysed in multiple myeloma.



Fig. 30.1: X-ray showing fracture lower end of radius and ulna.

Radioisotope bone scan using strontium (^{85}Sr) and radioactive $\text{Tc}^{99\text{m}}$ is very useful method to detect bone secondaries, aseptic necrosis, abscesses, arthropathies.

Arthroscopy is very useful to detect joint injuries by direct vision like in knee joint. **Arthrography** is injection of contrast into the joint and taking X-rays.

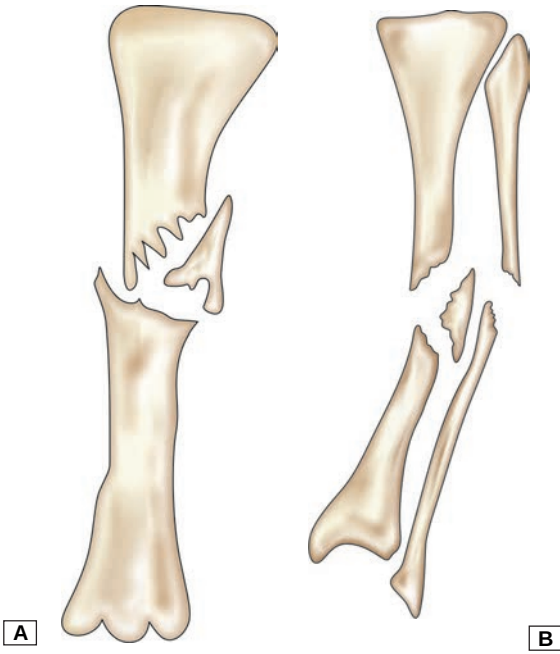
MRI is the investigation of choice for joint injuries. It clearly delineates the injuries.

Fracture

'Fracture' is defined as break in continuity of the bone.

Causes of fracture: *Trauma*—commonest cause. *Pathological*—It is due to underlying pathology like malignancy, secondaries in bone, osteoporosis, osteomyelitis, multiple myeloma, hyperparathyroidism, and rickets. *Stress fracture*—It is due to repeated minor trauma leading to repetitive stress to the bone causing fracture. It is common in second metatarsal of foot, occurs due to repeated marching and stamping. It is also called as '*March fracture*'. It is treated by rest, immobilisation with plaster slab of the foot.

Types of fracture: Given below a brief explanation of different types of fractures (Figs 30.2 to 30.6). *Greenstick fracture*—It is seen in children wherein bone breaks incompletely and partially keeping cortex intact. It is treated by rest, immobilisation. *Closed fracture*—Wherein fracture does not communicate outside. *Open fracture*—Wherein fracture communicates outside to skin through soft tissues exposing the bone, and allowing infection to get in. It is also called as '*compound fracture*'. *Transverse fracture*, *Oblique fracture*, *Spiral fracture* are other types of fractures based on fracture line. *Comminuted fracture*—Here bone is broken into more than two fragments. *Stellate fracture* begins at one point and radiates towards periphery as a star. It is common in patella and skull. *Avulsion fracture* occurs due to powerful contraction of muscle. *Depressed fracture* is common in skull. *Complicated fracture* is a fracture associated with injuries to vessels, nerves, joints.



Figs 30.2A and B: Comminuted fracture.

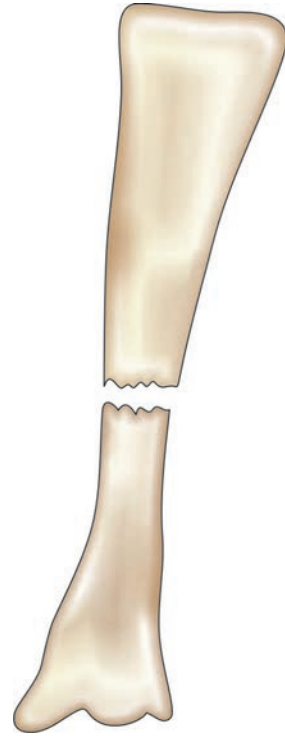


Fig. 30.4: Transverse fracture.

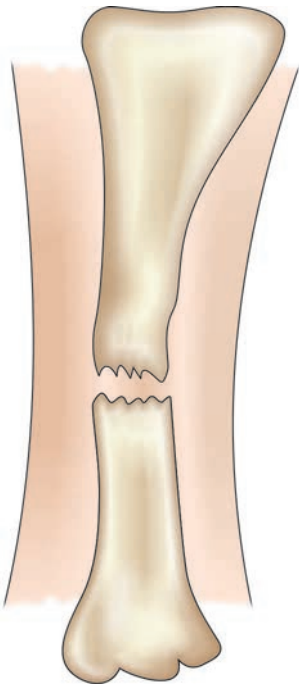


Fig. 30.3: Simple fracture.

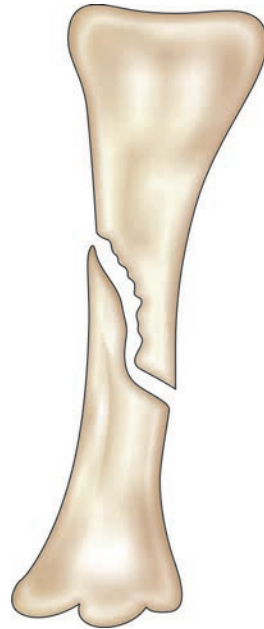


Fig. 30.5: Spiral fracture.

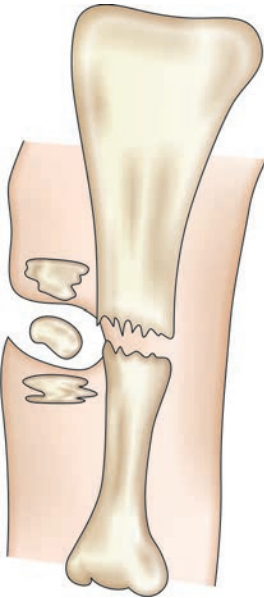


Fig. 30.6: Open fracture communicating externally.

Mechanism of injury in fracture

Direct violence
Indirect violence
Torsion forces.

Clinical features of fracture

Pain
Deformity
Swelling
Local bony tenderness
Shortening of the limb
Abnormal mobility
Crepitus
Loss of function
In fractures of femur and tibia, there will be features of shock.
Features of associated injuries.

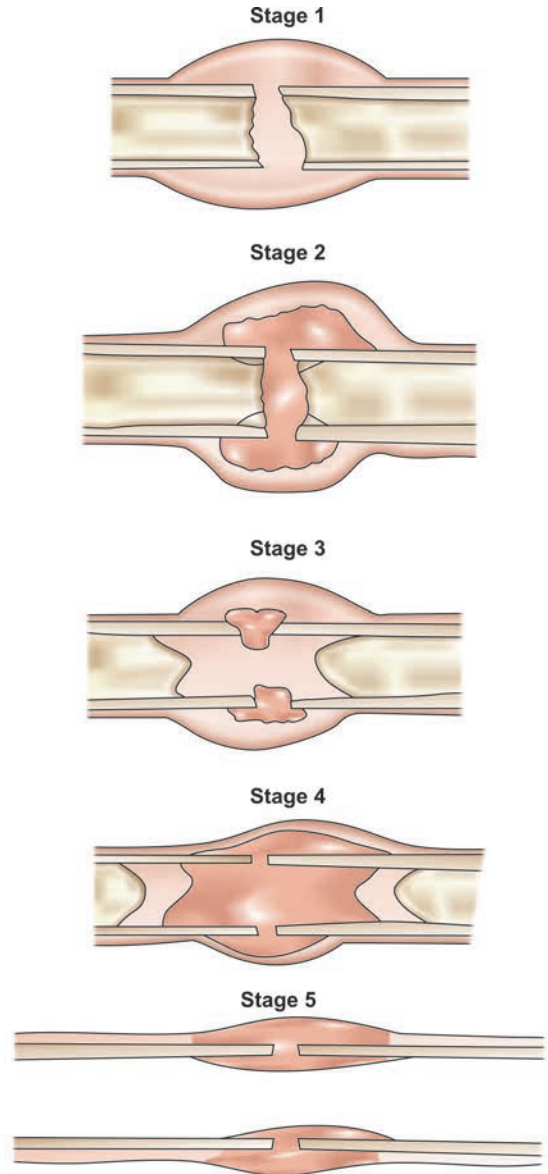


Fig. 30.7: Stages of healing of bone after injury.

Stages of healing of fracture (Fig. 30.7)

Stage of haematoma formation
Stage of granulation tissue formation
Stage of fibrocartilaginous callus
Stage of callus formation
Remodeling phase

Factors affecting fracture healing

Improper immobilisation
Infection
Interposition of soft tissues
Inadequate blood supply
Old age
Deficiencies of vitamin C, proteins
Anaemia
Diabetes, HIV, Steroid therapy

Complications of Fracture

Immediate—Shock; Injury to other structures; Compartment syndrome.

Delayed—**Fat embolism**—Due to fracture microscopic fat globules from the bone marrow enters the circulation and reaches the lung, brain and skin causing respiratory distress, drowsiness and petechial hemorrhage. Often it is life threatening.

Other complications are infection; Delayed nerve injury; Disability; Volkmann's contracture; 'Myositis ossificans', is common in elbow causing stiffness due to haematoma formation under the stripped periosteum—organisation—calcification in front of the elbow joint, common in children.

Late—**Malunion**—common in Colle's fracture; Nonunion; Osteomyelitis of the bone; Stiffness and contracture; Osteoarthritis of the joint, Sudeck's osteodystrophy, avascular necrosis (Bone will be denser on X-ray; occurs 3 months after fracture; common in fractures of neck of femur, scaphoid, neck of talus).

Nonunion: It is failure of bony union of the fracture segments even after consecutive three months of specified expected time of union. *Causes are*—infection; interposition of soft tissues; poor blood supply; wide separation of fracture segments; improper immobilisation. Site forms a pseudo joint with *painless abnormal mobility*. X-ray shows sclerosis of bone ends with a gap. It is common in—fracture of waist of scaphoid, neck of femur, neck of talus, and lower third of tibia. *Nonunion can be* hypovascular/avascular (true nonunion) or hypervascular types.

Delayed union: It is undue delay in the process of complete union. X-ray shows callus but inadequate. There is *painful abnormal mobility*.

Malunion: It is union of fracture in defective position with anatomical malalignment—angulation, rotation, overriding. It is due to improper reduction, redisplace-



Fig. 30.8: X-ray showing malunion in clavicle fracture. Note the site of fracture—lateral 1/3rd.

ment, and growth disturbance by epiphyseal injury. It is common in (cancellous bone) Colles', supracondylar (humerus), condyles of tibia fractures. *Features are*—deformity, no abnormal mobility; X-ray shows deformity with bridging callus (**Fig. 30.8**).

Sudeck's osteodystrophy (Paul Hermann Sudeck—German surgeon): It is post-traumatic painful osteoporosis with pain, swelling and marked stiffness of hand or foot of the injured limb. It may be due to abnormal activation of pain pathway with exaggerated inflammatory and immune responses. Features appear 2 months after trauma as—severe pain, swelling, hyperaemia, obliterated skin crease, glossy look, atrophied nails, impaired metacarpophalangeal and interphalangeal joint movements (Frozen hand) with marked stiffness. X-ray shows spotty osteoporosis with rarefaction.

Dislocation

Here one bony component of the joint completely loses its contact with other bony component. In subluxation, there is partial loss of contact between the joint surfaces. *Dislocation* can be traumatic; pathological; paralytic (poliomyelitis); congenital (Congenital dislocation of hip).

EXAMINATION OF JOINT INJURIES

Examination of Shoulder Joint in Injuries

Attitude: In anterior shoulder dislocation and fracture clavicle patient supports the flexed elbow of injured side with other hand.

Deformity: Swelling and prominences is seen in fracture clavicle. Abnormal swelling is seen in deltopectoral groove in subcoracoid dislocation (commonest type) with lowered anterior axillary fold, flattening and loss of roundness of the shoulder. Drooping of shoulder occurs in fracture neck of scapula.

Inspection

Inspection should be done from front, behind and sideward.

Palpation

Clavicle is palpated from behind beginning from sternal end towards acromion.

Upper end of the humerus is initially felt from behind with patient in sitting position. From the acromion process of both sides palpation is done using both hands downwards to feel greater tuberosity of humerus. It is not felt and there is no resistance in shoulder dislocation. Fingers are slowly slid downwards to feel the shaft of humerus for fracture. Bimanual palpation of the upper end of humerus is done from the side with one hand in the axilla and other over the shoulder. It is better felt through the axilla. Features of fracture should be looked for (**Figs 30.9 and 30.10**).

Palpation of scapula: First posteriorly subcutaneous portion of scapula is palpated, followed by vertebral border and inferior angle. Coracoid process is palpated 1.5 cm below the clavicle at the junction of medial 1/3rd and lateral 1/3rd. Glenoid cavity is palpated for tenderness. Coracoid process, acromial end of the clavicle and greater tuberosity are palpated for any deviation.

Arm length is measured from angle of acromion (scapular spine becomes acromion process) to lateral epicondyle. Length is increased in subglenoid dis-

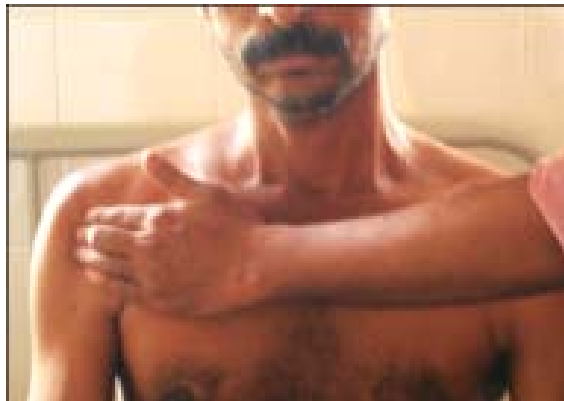


Fig. 30.9: Loss of roundness below the acromion is a feature of shoulder dislocation.



Fig. 30.10: Bimanual palpation of upper end of humerus across axilla and deltoid.

location and fracture scapular neck; it is shortened in subcoracoid dislocation (**Fig. 30.11**).

Vertical circumference of the axilla is measured for widening—*Callaway's sign*. *Bryant's test* is lowered anterior or posterior axillary fold.

Hamilton ruler test: A straight ruler is placed along the line of acromion process and lateral epicondyle. Normally it is not possible to place the ruler due to prominent greater tuberosity. In dislocation of shoulder it is possible as tuberosity is displaced medially.

Dugas' test: Patient is asked to keep the hand of the affected side on opposite shoulder. It is not possible if there is shoulder dislocation (**Fig. 30.12**).



Fig. 30.11: Length of the arm is measured from angle of acromion to lateral epicondyle.



Fig. 30.12: Dugas test—It is inability to place hand of the injured side on the opposite shoulder.

Sensation over the skin on the lower part of deltoid should be checked to find out axillary nerve palsy.

X-ray shoulder joint is essential to diagnose fracture/dislocation around the shoulder.

Fracture Clavicle

It is due to fall on the outstretched hand. Site is junction of lateral 1/3rd and middle third. Pain, tenderness, swelling are the features. It can be displaced or undisplaced.

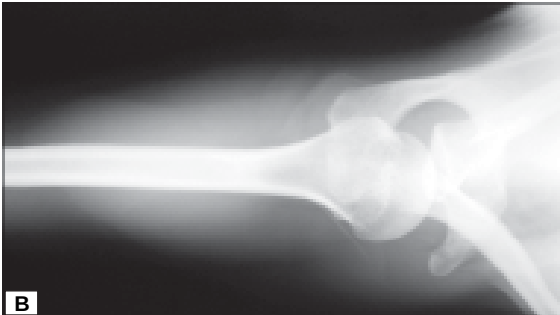
Shoulder Dislocation

It is common injury. It may occur after fall on outstretched hand. *Anterior subcoracoid dislocation* is common where head is located below the coracoid process. Occasionally head may lie below the glenoid cavity causing subglenoid dislocation. Very rarely posterior dislocation can occur in fully abducted arm with forcible internal rotation—*luxatio in erecta*.

Features (in subcoracoid): Flexed injured elbow is supported with normal hand by the patient. There is absence of round contour; flattening; bulge at the deltopectoral groove; patient cannot touch the opposite shoulder using diseased side hand (Dugas' test); Hamilton ruler test makes ruler easily to be placed. Anterior capsule of the joint is stripped off from the anterior margin of the glenoid rim causing recurrent dislocation of the head of the humerus within the capsule. Dislocation develops when patient abducts his arm at right angle and externally rotates it (**Figs 30.13A and B**).

Examination of Elbow Joint in Injuries

Attitude: Elbow is usually swollen, in flexed position. Normal outward deviation of extended supinated forearm (from axis of arm) is called *carrying angle* (10° - 15° ; more in women). It disappears in flexed and pronated elbow. It is increased in cubitus valgus; decreased in cubitus varus. Olecranon becomes unduly prominent with anteroposterior broadening of the elbow in supracondylar fracture and posterior dislocation of elbow (**Fig. 30.14**).



Figs 30.13A and B: X-ray in two planes to be taken in shoulder dislocation.

Swelling, deformities are to be examined. Local bony tenderness, irregularity; undue mobility; crepitus should be checked. All bones around the elbow and 3 bony points (two epicondyles, olecranon) should be examined. Lower third of the humerus is examined for supracondylar fracture in children, T/Y fractures in adults.

Upper end of the radius is felt in flexed elbow just below the lateral humeral condyle while forearm is pronating and supinating. Fracture head (adult) or neck (children) of radius is checked. Squeezing the radius

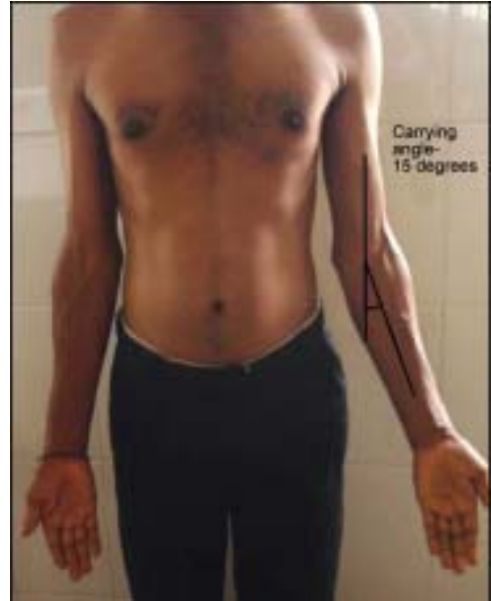


Fig. 30.14: Normal carrying angle is 15° outwards in an extended supinated forearm. It disappears on flexion.



Fig. 30.15: Head of radius is felt by pronating and supinating the forearm in flexed elbow.

and ulna by holding together at lower end of the forearm will cause pain in the upper end of radius—*springing the radius*. Upper end of the ulna should be examined (**Figs 30.15 and 30.16**).

Relative positions of medial and lateral epicondyles and olecranon should be checked—by placing thumb and middle fingers on epicondyles and index finger on the olecranon. It forms a straight line in extended position; but forms a triangle in flexed position with shortest part is between medial epicondyle and olecranon and longest part is between epicondyles. Triangle is useful in assessing posterior dislocation, T/Y fractures (**Figs 30.17A and B**).



Fig. 30.16: Index is used to feel lateral epicondyle and middle finger is used to feel head of radius.



A



A

Figs 30.17A and B: Three bony points form a straight line in elbow when extended; form a triangle in elbow when flexed.

Measurements: Forearm should be at right angle to arm while measuring. Forearm is measured from lateral epicondyle of humerus to radial styloid process.

Movements: Flexion is around 145°-160°. Flexion and extension should be checked against resistance. Flexion is by brachialis, biceps, brachioradialis; extension is by triceps, anconeus. Patient is asked to touch his shoulders with the flexed elbows to check active flexion.

Pronation and supination occurs in vertical axis from head of radius to ulnar side of articular disc of radius. In pronation hand faces downwards; in supination hand faces upwards (King pronates, beggar supinates). Supination is antigravity and is powerful. Normal pronation is 70°; supination is 85°. Both are checked in flexed position with arm beside the patient.

Always check the radial pulse and neurological status of all three nerves—radial, ulnar and median.

Supracondylar fracture: Backward displacement due to fall on outstretched hand with flexed elbow is common type. Distal fragment displaces backward, upward, with internal rotation. Forward displacement is rare, due to fall on outstretched hand with extended elbow. *Complications:* Malunion, cubitus valgus/varus, injury to brachial artery or nerves, myositis ossificans, Volkmann's ischaemic contracture (**Fig. 30.18**).



Fig. 30.18: Abnormal mobility is checked in supracondylar fracture.

Posterior Dislocation of Elbow

It occurs due to fall of outstretched hand with slightly flexed elbow. Fracture of coronoid process and lateral displacement are common associations.

Features: Common in adult; shortening of forearm; posterior displacement of olecranon; absence of abnormal mobility and crepitus. Subluxation of head of radius is seen in children while pulling the forearm in supinated position (pulled elbow). Head of radius is below and lateral than normal.

Monteggia Fracture Dislocation

It is fracture of upper third ulna with anterior displacement of upper fragment of ulna and anterior dislocation of radius. In *reversed Monteggia fracture dislocation* there is posterior upper ulnar fracture displacement and anterior dislocation of radius. High Monteggia fracture is *Hume's fracture*.

Other fractures are—T/ Y fracture, fractures of lateral/medial epicondyle, capitulum, neck/head of radius, olecranon process.

Examination of Wrist in Injuries

Deformities and attitude: *Dinner fork deformity* is a feature of Colles' fracture. An abnormal slight anterior projection of the wrist is due to dislocation of lunate bone. Different conditions show different deformities in wrist, hand and fingers.

All features typical of any fracture should be looked for.

Lower third of radius, lower third of ulna should be examined. It is important in Colles', Smith's, Galeazzi fractures. Tenderness, irregularity, displacement, crepitus should be checked.

Level of two styloid processes: Normally radial styloid process is about half an inch below the ulnar styloid process. Patient keeps his forearm pronated, index fingers of the examiner are placed over the styloid process to demonstrate the level. In Colles' fracture radial styloid is at same level or higher than ulnar styloid (**Fig. 30.19**).

Scaphoid is palpated at anatomical snuff box with the wrist bent medially. Tenderness may suggest fracture.



Fig. 30.19: Tip of radial styloid process is 1 cm lower than tip of ulnar styloid process.

Movements: Wrist movements (refer Chapter 27: Examination of Hand and Foot), finger, thumb movements should be checked carefully.

X-ray is diagnostic. For scaphoid (Skaphos—boat) fracture oblique view is essential. Often fracture may be identified only when X-ray is repeated after 10 days. Often MRI may be needed to identify scaphoid fracture.

Colles' Fracture (Sir Abraham Colles' 1814)

It is fracture radius at 2 cm proximal to the distal articular surface of the radius with dorsal, lateral, proximal displacement of distal fragment—*dinner fork deformity*. It occurs following a fall on an outstretched hand; common in elderly postmenopausal osteoporotic women.

Features: Deformity; swelling; irregularity; loss of normal concavity of radius; radial styloid process from its normal lower position becomes equal or higher than ulnar styloid process.

Complications: Stiffness; malunion; manus valgus; Sudeck's osteodystrophy; causalgia; rupture of extensor hallucis longus tendon, carpal tunnel syndrome (**Fig. 30.20**).

Smith's Fracture

It is a true reversed Colles' fracture with transverse fracture of the lower end of radius 2 cm proximal of the articular surface with anterior displacement—*garden spade deformity*.



Fig. 30.20: Colles' fracture.

Chauffeur's Fracture

It is fracture radius just above the styloid process used to occur when crank used to start the car kicked back.

Madelung Deformity (Otto Madelung, France 1926)

It is dorsal subluxation of lower end of ulna seen in young girls with weak wrist; unstable inferior radio-ulnar joint; may be congenital or acquired; probably due to delayed growth of radius as a result of repeated trauma but continuous growth of ulna forces ulna to subluxate dorsally causing prominent dorsum of the wrist.

Scaphoid Fracture

It is due to fall on outstretched dorsiflexed radially deviated hand in young individual due to impact of scaphoid against radial styloid. Fullness and tenderness with medially deviated wrist are the features. Oblique scaphoid view X-ray or MRI is needed. It often needs to repeat X-ray after 10 days.

Complications: Nonunion, avascular necrosis of proximal part, osteoarthritis of wrist.

Lunate Dislocation

It is due to fall with outstretched hand where entire carpus displaces backwards. Lunate bone is displaced anteriorly and rotated 90° horizontally. In perilunate dislocation remainder of carpus is separated from the lunate bone and so entire carpus is dislocated posteriorly except lunate.

Complications: Median nerve injury; avascular necrosis (Kienbock's disease), osteoarthritis.

Bennett's Fracture Dislocation

It is an oblique fracture occurring at the base of 1st metacarpal bone with subluxation of the carpometacarpal joint. Triangular fragment of fractured bone remains in position. If fracture is T or Y shaped it is called as *Rolando's fracture*.

Galeazzi Fracture

It is fracture of lower end of radius (1/3rd or 1/4th) with dislocation or subluxation of inferior radioulnar joint. It is due to rotational force causing swelling lower forearm; prominent head of ulna; ulnar nerve injury.

Fractures occurring due to fall with outstretched hand

- Fracture of clavicle
- Fracture neck of humerus
- Fracture shaft of humerus
- Supracondylar fracture of humerus
- Posterior dislocation of elbow
- Fracture of radius and ulna
- Colles' fracture
- Scaphoid fracture

Injuries to Pelvis

Pelvis gets injured in crush injury, road traffic accidents, direct or indirect violence. Shock, severe pain, inability to move pelvis is typical. External rotation force, compression force, vertical shear are the type of forces which cause pelvic injury.

Ecchymoses, bruising, swelling in lower abdomen, perineum, scrotum and penis are common. Associated injuries of limbs, abdomen, and thorax also should be confirmed. Urethral/rectal/vaginal bleeding is common. A large haematoma in the inguinal region or scrotum is called as *Destot's sign*. Decrease in distance between the greater trochanter and pubis is called as *Roux sign*. On rectal examination a tender bony part or large haematoma may be felt and is called as *Earle sign*.

Stability of the pelvis should be checked by compressing two iliac bones (**Fig. 30.21**).



Fig. 30.21: Two iliac bones are compressed inwards to check the stability and tenderness in pelvis.

Isolated Pelvic Injury

It is undisplaced ischial or pubic fractures that do not disrupt the integrity of pelvic ring. It commonly occurs over superior or inferior ischiopubic ramus or both rami. Here problems are less with minimum complications.

Fracture with Disruption of the Pelvic Ring

Here fracture occurs at two opposite points with separation and disruption. Anterior injury with fractures through both ischiopubic rami with separation/disruption of pubic symphysis is common type.

Avulsion Fractures

Sartorius (from anterior superior iliac spine); rectus muscle (anterior inferior iliac spine); hamstring (ischial tuberosity).

Injury to Sacrum and Coccyx

Injury to sacrum and coccyx also can occur presenting with severe pain.

Malgaigne Fracture

It is fracture through rami or symphysis pubis with disruption of posterior arch through sacrum/sacroiliac joint/ilium causing unstable anterior as well as posterior arch failure.

Complications: Paraesthesia of lower limb; severe limp; severe back/groin pain; pelvic obliquity; neurologic problems, changes in leg length.

Complications of Pelvic Injury

Rupture of bladder; rupture of the urethra with extravasation of urine; injury to rectum; injury to major vessels causing torrential haemorrhage; injury to lumbosacral plexus nerves; acetabular fracture with osteoarthritis. *CT pelvis is a must.*

Examination of Hip in Injuries

Fracture femoral neck, dislocation of head of femur are the common injuries. Accidents, fall, impact on the greater trochanter may force the head across acetabulum causing posterior dislocation. History of pain, inability to get up, deformity, are typical. Patient may get up and stand with pain if it is an impacted fracture. Fracture neck can develop even in minor injury in elderly.

Attitude: Externally rotated lower limb is due to fracture neck of femur (in elderly). Flexed, internally rotated, adducted attitude is seen in posterior dislocation of hip (in young). External rotation, abduction, flexion is seen in anterior dislocation which is rare (**Fig. 30.22**).

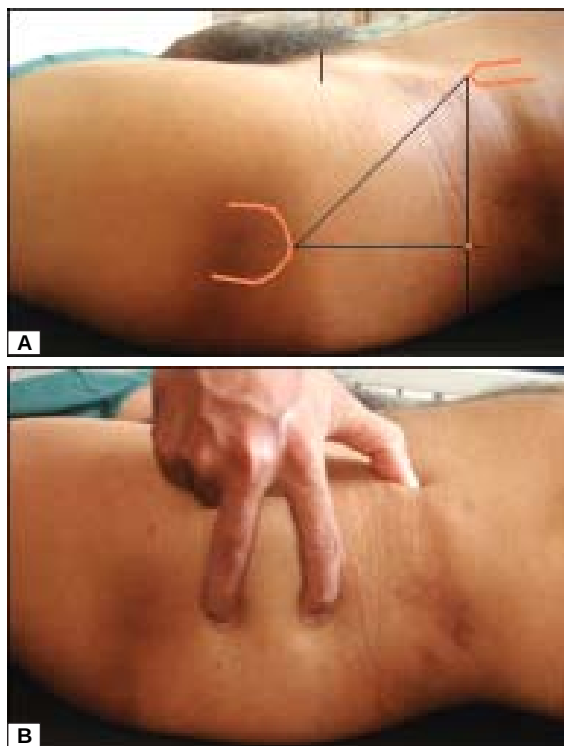
Bruising, swelling, haematomas are observed.

Position of head of femur: It is in the dorsum ilium in posterior type; in the groin in pubic type; in the perineum in obturator type. Femoral artery which is normally felt over the head of femur is not felt in dislocation—*Narath's sign*. Tenderness during rotation of femur or over trochanter may be felt.



Fig. 30.22: External rotation of hip (left side) is feature of fracture neck of femur.

Bryant's triangle: Vertical downward line is drawn first from anterior superior iliac spine; second line is drawn from tip of anterior superior iliac spine to tip of greater trochanter; third horizontal line is drawn from tip of trochanter to first line. Short segment of this triangle is the third line. Upward elevation of trochanter reduces the length of this third line. Second line is increased in anterior dislocation; reduced in posterior dislocation (**Figs 30.23A and B**).



Figs 30.23A and B: Method of drawing Bryant's triangle and feeling its points.

Nelaton's line: With patient lying down on his sound side, a line is drawn from ischial tuberosity to tip of anterior superior iliac spine. In normal individual this line touches the tip of greater trochanter (**Fig. 30.24**).

Schoemaker's line: It is the extended line from greater trochanter to anterior superior iliac spine to reach the umbilicus. Line shifts below the umbilical level if greater trochanter is elevated.

Chiene's test: A tape joining the tips of greater trochanters is parallel to another tape joining the two



Fig. 30.24: Nelaton's line.

anterior superior iliac spines. Two will converge towards diseased side when trochanter is elevated.

Morris bitrochanteric test: Using pair of calipers distance between outer margin of greater trochanter and pubic symphysis is compared. There is medial displacement of trochanter in posterior and central dislocation and lateral displacement in anterior dislocation.

Measurements: Lower limb is measured after placing the sound limb in identical position as diseased limb. Pelvis should not be tilted and two anterior superior iliac spines should be in the same line. Lower limb is measured from anterior superior iliac spine to medial malleolus. Thigh is measured from anterior superior iliac spine to knee joint line in flexed knee position. In all fractures and dislocations there are shortening except in obturator type of dislocation.

Movements of the hip joint should be checked. Normal flexion is 110° ; extension is 30° from neutral; abduction is 50° ; adduction is 30° ; internal rotation in flexion is 45° ; external rotation in flexion is 45° ; external rotation in extension 45° ; internal rotation in extension is 35° .

Rectal examination reveals palpable head of femur in central dislocation.

Complications in hip injury: Bleeding; sciatic, femoral, obturator nerve injuries.

X-ray hip is very useful. *Pauwel's angle* formed by fracture line to horizontal plane; position of lesser trochanter, Shenton's line; acetabulum are the points to be assessed.

Fracture Neck of Femur

It is common in elderly and females. It can be—subcapital (70%); basal (10%); transcervical (20%). It can be—incomplete; complete without displacement; complete with minimum displacement; complete with marked displacement.

Features: Flexion, abduction and external rotation is the attitude with tender midinguinal point; shortening.

Complications: Avascular necrosis; nonunion; osteoarthritis.

Dislocation of Hip

Posterior dislocation is commonest type. It causes flexion, adduction, and internal rotation with shortening, impalpable femoral artery pulse (*Narath's sign*). *Anterior dislocation* occurs while stepping into a boat with abducted limb causing flexion external rotation and abduction. Head is adjacent to pubic symphysis (*pubic type*) or under adductor muscles (*obturator type*). Obturator type causes lengthening of the limb. In *central dislocation* head of femur is forced through fractured acetabulum into the pelvis making it to palpate per rectally (**Fig. 30.25**).

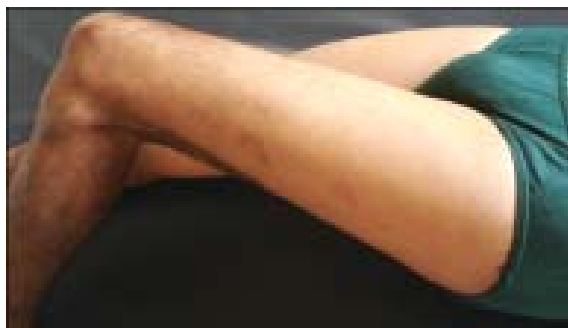


Fig. 30.25: Posterior dislocation of left hip.

Avulsion Fracture of Lesser Trochanter

It is due to violent contraction of the iliopsoas muscle. *Ludloff's sign:* Inability to flex the stretched leg once patient is seated.

Intertrochanteric Fracture

It is extracapsular fracture between greater and lesser trochanter of femur. It unites readily (cancellous bone) with less complications.

Features: Marked pain, tenderness, ecchymoses and haematoma over the trochanteric region with abduction, external rotation and shortening. It can be of undisplaced/displaced two fragments or three fragments or four fragments or reverse oblique.

Subtrochanteric Fracture

It is fracture of femur occurring upto 5-7.5 cm below the lesser trochanter. It is difficult fracture as it is in cortical bone; it is an area with greater stress and force. Proximal segment is flexed (iliopsoas), abducted (gluteus medius); distal segment is adducted (adductors). Bleeding and haematoma is common (**Fig. 30.26**).



Fig. 30.26: Shaft of the femur when moved greater trochanter will be immobile in subtrochanteric fracture.

Examination of Knee in Injuries

Attitude: Knee commonly gets locked in flexed position. It can often be abducted, adducted, hyperextended.

Swelling: It could be due to effusion, haemarthrosis causing typical horse shoe look—around superior and lateral aspect of the patella. Wasting of quadriceps some time is common after knee injury. Patellar dislocation is towards lateral side causing swelling laterally.

Medial collateral ligament injury causes tenderness at its femoral attachment. Medial semilunar cartilage causes tenderness at joint level without any bony tenderness. Injury to anterior horn of medial semilunar cartilage causes tenderness between ligamentum patellae and medial collateral ligament (it is elicited on flexed knee using thumb pressing at this point and knee is gradually extended). Tear of its posterior horn causes tenderness behind medial collateral ligament.

Patella is palpated for tenderness, gap, irregularity (bruising over the surface is common). Injury to

extensor mechanism of knee (quadriceps, patella, ligamentum patellae) makes patient unable to lift the extended leg. Patellar tap should be checked.

Lower end of femur in supracondylar fracture will be directed backward and upper end will be directed forward. Upper end of tibia and fibula should be palpated carefully. Lateral tibial condyle is commonly fractured than medial. Squeezing the lower part of tibia and fibula will cause tenderness in fibular fracture site (upper end of fibula—*springing of fibula*) (Fig. 30.27).



Fig. 30.27: Springing of fibula.

McMurray's test: Knee joint is flexed, foot is held and *externally rotated*; leg is *abducted* and knee slowly is extended. Patient develops *pain and click* due to injury to *medial semilunar cartilage*. Pain and click if develops in the beginning means injury to posterior part of medial semilunar cartilage, in the middle of extension means injury to middle of cartilage; at the end of extension means injury to anterior end of cartilage. *Lateral semilunar cartilage* is checked similarly by *internal rotation* of foot and *adduction* of leg (Fig. 30.28).



Fig. 30.28: McMurray's test.

Apley's grinding test: With the patient in prone position, examiner keeps his knee on patient's thigh and patient's knee is flexed and foot is held to rotate the leg externally with compression and grinding to elicit the pain in case of medial semilunar cartilage injury. Foot is rotated internally to elicit pain in case of lateral semilunar cartilage injury (Figs 30.29A and B).



Figs 30.29A and B: Apley's grinding test.

Pulling the leg upwards with external rotation causes pain in medial collateral ligament injury (common). If internal rotation causes pain then there is injury in lateral collateral ligament (Rare)—*Apley's distraction test*.

Abduction and adduction tests for stability: With one hand foot is held up with fully extended knee, other hand is kept at knee; leg is abducted to feel the abnormal opening of the joint on the medial side in case of medial collateral ligament injury; if adducted joint will open laterally in lateral collateral ligament injury (Fig. 30.30).

Drawer sign: With knee flexed (right angle) and foot resting on the bed, foot is fixed with one hand or by sitting on foot, upper end of tibia is moved with the other hand anteroposteriorly to check increased mobility. Increased anterior mobility means it is anterior cruciate ligament injury; increased posterior movement means it is injury of posterior cruciate ligament (Figs 30.31 and 30.32).

Movements of knee: Flexion is normally 135°. Extension, abduction and adduction are checked.

Hip, ankle joints should be examined.



Fig. 30.30: Abduction of knee to confirm medial collateral ligament injury.



Fig. 30.31: Drawer sign. Forward push is done to check anterior cruciate ligament; backward push for posterior cruciate ligament.



Fig. 30.32: Checking the stability of cruciate ligaments by moving upper end of tibia on femur.

Complications: Popliteal artery injury, venous oedema, haematoma calf, compartment syndrome, nerve injuries (lateral peroneal nerve in fibular injury) can occur.

Patellar fracture: It occurs by direct or indirect (Muscular violent contraction) trauma. Direct trauma results in comminuted fracture; indirect in transverse fracture. Swelling, effusion or blood in knee joint, gap, irregularity in patella are the features (**Fig. 30.33**).



Fig. 30.33: Patellar fracture.

Medial collateral ligament is commonly injured than lateral. Distraction test, abduction test, pain and tenderness, instability are the features.

Medial semilunar cartilage (meniscus) is commonly injured than lateral as it is fixed to medial collateral ligament. It can be *anterior* horn tear or *posterior* horn tear or *bucket handle* tear. Joint gets locked and later suddenly gets unlocked and gives way. Joint line tenderness, effusion, McMurray's and Apley's grinding tests are positive.

Cruciate ligament injuries: It is due to severe trauma. It causes joint instability confirmed by Drawer's sign. Hyperextension of knee is common in anterior cruciate ligament injury.

Pellegrini-Stieda's disease is calcification of medial collateral ligament after partial avulsion from medial femoral condyle.

Loose bodies in knee joint**Types**

Fibrous—traumatic, tuberculosis, syphilis, osteoarthritis

Fibrinous—traumatic, tuberculosis, chronic synovitis

Cartilaginous—meniscal injury

Osteocartilaginous—osteochondritis dissecans, osteophytes detached, sequestrum, synovial chondroma, fracture tibial spine

Others—foreign body, lipoma, carcinoma

Diagnosis—locking; X-ray, arthroscopy, MRI

Examination of Ankle in Injuries

Three types of injuries occurs in ankle—external rotation; internal rotation; vertical compression. Slip, violence, twist, crush, accidents can cause ankle injuries.

Displacement laterally or medially; front or behind; broadening of ankle; swelling; bruising; open wound exposing the bones; shape; posture should be observed. All bony parts around ankle—malleoli, lower ends of fibula and tibia, calcaneum, talus, tarsal bones, metatarsals should be palpated.

Tendoachilles should be examined.

Measurements: Distance between malleoli and point of heel; distance between medial malleolus to head of first metatarsal bone and point of heel; distance between the lateral malleolus to the head of the 5th metatarsal bone and point of heel are measured. These measurements give the idea about the displacements.

Movements: *Dorsiflexion* at ankle is 25° ; *plantar flexion* at ankle is 50° . Left hand is placed behind the lower leg; right hand is placed over hindfoot (not over forefoot as movements of midtarsal and subtalar joints are to be eliminated). Dorsiflexion and plantar flexion is checked with knee slightly flexed and heel off the ground. *Inversion and eversion* is checked at *subtalar* joint (between talus and calcaneus). Lower leg is supported by holding the ankle firmly with one hand; with other hand calcaneum is grasped to check the inversion and eversion. Normal range is of 20° . *Abduction and adduction* occurs in *midtarsal* joint (between hindfoot and midfoot—Chopart's). Calcaneum is held firmly with one hand; midfoot is grasped near bases of metatarsals to elicit abduction and adduction. Normal range of each is 20° . **Note:** *Forefoot* is metatarsals and phalanges; *midfoot* is navicular, cuboid, 3 cuneiforms; *hindfoot* is calcaneum

and talus. *Subtalar joint* is between talus and calcaneum. *Midtarsal joint* (Chopart's) is between hindfoot and midfoot. *Lisfranc's joint* is between midfoot and forefoot (**Figs 30.34 to 30.37**).

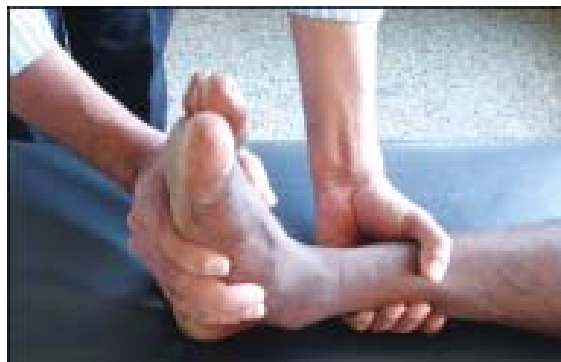


Fig. 30.34: Eliciting ankle joint movements—dorsiflexion and plantar flexion.



Fig. 30.35: Eliciting subtalar joint movements—inverson and eversion.



Fig. 30.36: Eliciting midtarsal joint movements—abduction and adduction.



Fig. 30.37: Normal dorsiflexion of great toe is 90°.

X-ray foot in different views is necessary. *Tuber—joint angle* formed by a line passing over the non-articular surface of the calcaneum and another line over the articular surface of talus which is normally 40°. MRI foot is often needed.

Injuries around ankle: *External rotation injury* causing spiral fracture of fibula, avulsion of medial malleolus, avulsion of posterior fragment of tibia with tibiofibular diastasis. *Abduction injury:* Transverse fracture of fibula 5 cm above ankle, avulsion of medial

malleolus. *Adduction injury:* Vertical fracture of the medial malleolus, avulsion of tip of fibula.

Chopart's injury: It is fracture dislocation of midfoot involving calcaneum, talus, navicular bones.

Lisfranc's injury: It is fracture dislocation between midfoot and forefoot involving metatarsals, often cuneiform, cuboid bones. It is a complex complicated fracture.

Jones' fracture is a transverse fracture of base of 5th metatarsal bone by indirect violence.

Other fractures: Isolated fracture of medial malleolus (Tillaux); fracture lateral malleolus with lateral displacement of talus; fractures of both malleoli with displacement of talus (Pott's fracture); diastasis of inferior radioulnar joint (Dupuytren's fracture); posterior marginal fracture of tibia (Trilamellar fracture/Cotton fracture); vertical compression fracture of tibia.

Calcaneal fracture (Lover's fracture/Don Juan fracture): It is usually due to fall from height. It can be split or crush (compression) fracture. It can be undisplaced or displaced. Often it may be associated with compression fracture of spine. So spine should be examined in these patients.

EXAMINATIONS IN BONE DISEASES

History

Age of onset of disease: Secondaries in bone, Paget's disease of bone occur in old age; multiple myeloma between 40-50 years; osteoclastoma 30-40 years; osteosarcoma 15-30 years; benign tumours of bone, fibrous dysplasia occurs in adolescents; solitary bone cyst, acute osteomyelitis, osteogenesis imperfecta tarda occurs in children; osteogenesis imperfecta congenita occurs at birth.

Trauma may be the cause for osteomyelitis. Acute onset is usually due to acute osteomyelitis. Bone tumours develop spontaneously. Rapidly growing swelling may be osteosarcoma.

Pain is a feature of acute osteomyelitis. Dull aching pain may develop later in malignant tumour. But in osteosarcoma pain may be the initial presentation.

Short duration disease of bone may be acute osteomyelitis or malignancy.

History of discharging sinus (chronic osteomyelitis), its duration, and content should be asked. Similar swellings in the body (osteochondroma) should be asked.

Earlier history of ear/skin/respiratory infections or typhoid or tuberculosis should be asked.

Family history of bone diseases like achondroplasia, diaphysial aclasis, Marfan's syndrome.

Examination

Swelling arising from bone is always *fixed* to bone and is nonmobile.

Inspection

Skin: Red congested oedematous skin is seen in acute osteomyelitis; tense, shiny, with dilated veins in osteosarcoma; multiple sinuses with sprouting granulation in the skin are due to chronic osteomyelitis or with undermined edge in tuberculosis; multiple healed scars suggest previous osteomyelitis.

Limb oedema, wasting of muscles, joint above and below should be examined. Joint effusion is common. Valgum, varus deformity can develop due to destruction of epiphyseal cartilage. Limb shortening or lengthening can occur.

Swelling for different features should be noted.

Palpation

Local rise of temperature is seen in osteomyelitis and osteosarcoma (due to increased vascularity). Tenderness is a feature of inflammation.

Swelling is always fixed to bone. Its location, size, shape (diffuse is inflammatory, pedunculated is exostosis, localised spherical may be bony tumour), surface (smooth in benign tumour, irregular in malignancy and chronic inflammation), edge (ill-defined in inflammation, well-defined in tumours) and consistency (bony hard in osteoma, egg shell crackling in osteoclastoma, variable in osteosarcoma). Highly vascular osteosarcoma, aneurysmal bone cyst, bone haemangioma, vascular secondaries (from follicular carcinoma of thyroid) are often pulsatile. It should be auscultated for bruit also.

Detailed palpation of ulcers or sinuses should be done.

By palpating the bony surface using thumb (gently run the thumb on the surface) gap due to pathological fracture can be made out.

Distal pulses, neurological examination (sensory, motor), muscle power distally and proximally should be checked.

Measurements: Shortening occurs in epiphyseal destruction. Hyperaemia in metaphysis causes lengthening. Muscle wasting is confirmed by measuring circumference and comparing to opposite side. Wasting is common in tuberculosis.

Movements of joints above and below should be checked. Gait should be checked.

Other bones in the body should be examined (Multiple exostoses, secondaries).

Drainage lymph nodes should be palpated. Its number, size, surface, consistency, fixity should be checked.

Systemic examination: Lungs should be examined for pulmonary tuberculosis or secondaries; abdomen should be examined. Site for primary (thyroid, kidney, breast, lungs, prostate, testis, GIT) should be looked for if bony lesion is suspected to be due to secondaries.

Epiphysis	Metaphysis	Diaphysis
Epiphysitis	Acute osteomyelitis	Syphilis
Osteoclastoma	Brodie's abscess	Ewing's tumour
	Tuberculosis	Multiple myeloma
	Osteoma	
	Chondroma	
	Osteosarcoma	
	Bone cyst	

Investigations

Blood: Total count (osteomyelitis), ESR, serum calcium (hyperparathyroidism, secondaries, sarcoidosis, myeloma), alkaline phosphatase, acid phosphatase, serum phosphorus, serum globulins are done in different situations.

Urine for albumin, Bence Jones proteins.

X-ray: It is not useful in acute osteomyelitis up to 10 days of onset of disease. It is useful in chronic osteomyelitis, all bone tumours, Paget's disease, Rickets, myeloma, hyperparathyroidism. Chest X-ray is needed to see lung secondaries.

Angiography is needed in malignancy and Paget's disease of bone.

Open biopsy is ideal for bone tumours. FNAC is done only in spine tumours.

Bone marrow study is needed in multiple myeloma for plasma cells.

Discharge for culture, AFB is necessary.

Radioactive bone scan: It is used in infective as well as neoplastic conditions. 10 mCi of ⁹⁹Tc-m-phosphate is injected intravenously. Scan is done in 4 hours (using

gamma camera) to get the picture. Bladder should be empty while scanning pelvis. It is commonly used in assessing bone secondaries.

Infections of the Bone

Bone gets infected by gram positive and gram negative bacteria, tuberculosis, brucellosis, typhoid, syphilis. Infection of the bone along with bone marrow is called as *osteomyelitis*. It can be acute or chronic.

Acute Osteomyelitis

It is acute bacterial infection of the bone.

Causes: Gram positive bacteria like staphylococci, streptococci, gram negative bacteria like *Klebsiella*, *Pseudomonas*.

Mode of infection: Direct—through the traumatic wounds; Haematogenous—from an infective focus from different parts of the body, like skin infections.

Mode of spread: Bacteria spread through metaphyseal vessels into the metaphysis of the bone and multiply there releasing toxins and evoking inflammation. These vessels being end arteries get compressed by increased pressure in the metaphysis causing pus formation and suppuration. This pus can spread longitudinally into the diaphysis or across the joint capsule into the joint causing arthritis. It can spread outward into the soft tissues [muscles and fascia] and reach the subcutaneous plane causing a swelling. It eventually bursts through the skin forming discharging sinuses. Periosteum lays new bone eventually, as a reaction.

Clinical features: Common in young boys; presents with fever, toxicity, pain in the bone, tender swelling at the site, effusion in adjacent joint, inability to move the limb.

Investigations: X-ray shows no changes for initial 2 weeks. Later it shows widening of cortical margin with new bone formation. Often joint effusion may be visualised. Total count and ESR may be increased. Other investigations done are blood culture. MRI bone.

Sequelae: Septicaemia, pyogenic arthritis, chronic osteomyelitis; limb shortening, disability; recurrent infection; chronic discharging sinus formation.

Chronic Osteomyelitis

It is chronic, recurrent bacterial infection and inflammation of bone and bone marrow usually of long bones. There is new bone formation called as *involucrum* (like envelope) with discharging sinus with bone spicules in the sinus. It occurs either due to trauma or as sequelae of acute osteomyelitis. Dead part of the bone within the infected bone is separated by granulation tissue and is called as *sequestrum* (Dead bone *in situ*) (**Figs 30.38 to 30.40**).

Clinical features: Discharging sinus with swelling; bone pain and tenderness; thickening of the bone; shortening, restricted mobility and deformity.

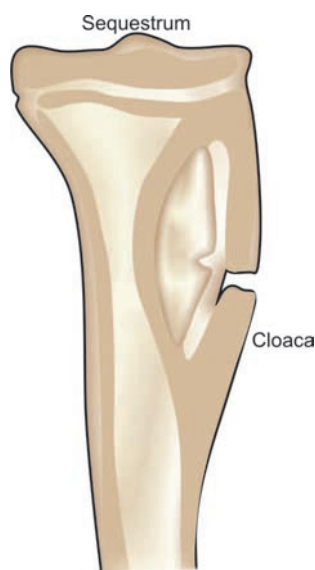


Fig. 30.38: Diagram showing osteomyelitis.



Fig. 30.39: Osteomyelitis leg and foot with multiple discharging sinuses.



Fig. 30.40: X-ray showing chronic osteomyelitis of femur.

Problems: Deformity; malignant change—Squamous cell carcinoma; Amyloid deposition; Recurrence.

Pus can get localised in the metaphysis to form an abscess called as **Brodie's abscess**. It can cause pain, tenderness and swelling in the bone. It can lead to pathological fracture. It requires open drainage and curettage. It is common in upper end of tibia and humerus.

Pathological States of Bones

Paget's Disease of Bone

It is also called as *Osteitis deformans* wherein there is increased blood supply to the bone, causing bone to enlarge more than normal because of more vascularity (vascular stage) and laying of coarse fibred abnormal bone (sclerotic stage). This is thick but not strong and is vulnerable for pathological fracture. Common bones involved are long bones like femur and tibia, spine, skull. This bone is more prone to develop osteogenic sarcoma.

Clinical features: Common in elderly male; Thickening and increase in visible size of the bone like in skull causing requirement of larger sized caps; Dull continuous pain in the bone with often pathological fracture; Increased vascularity in bone causes

hyperdynamic circulation leading to congestive cardiac failure; Bending (bowing) of leg bones. Paraplegia due to vertebral involvement; Deafness due to middle ear sclerosis; Involved bone is more prone for osteosarcoma than normal bone.

Investigations: Raised serum alkaline phosphatase; Elevated urinary excretion of hydroxy-proline; X-ray shows dense sclerotic bone; bone scan confirms the diagnosis (**Fig. 30.41**).

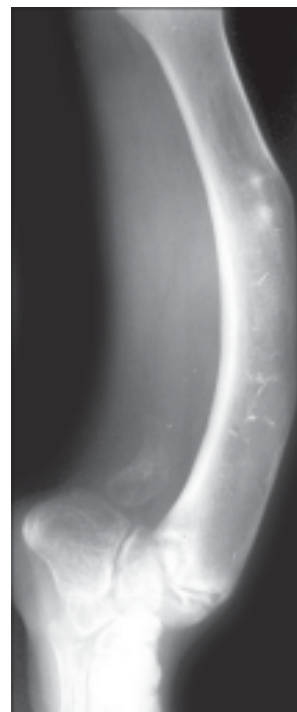


Fig. 30.41: Paget's disease of bone—tibia.

Rickets

Formed bone matrix is not calcified leading to rickets. Bones contain uncalcified bone matrix called as *osteoid*.

Causes: Dietary deficiency of vitamin D prevents calcium absorption from the gut. Excessive excretion of calcium in the kidney leads to less available calcium for bone calcification—*Renal rickets*.

Clinical features: Seen in infants; *Ricketic rosary* in costochondral junction; Bosselated frontal bone; Tri-radiate pelvis; Bowing of long bones; Stunted

growth; X-ray confirms the disease; Serum calcium and serum phosphatase level estimation.

Osteomalacia is vitamin D deficiency seen in adults (features are like rickets). Treatment is also same.

Bone Disease of Hyperparathyroidism (von Recklinghausen's Disease of Bone)

Increased parathormone secretion by hyperfunctioning parathyroid glands or by parathyroid adenoma causes calcium resorption from the bone replacing the bone with fibrous tissue often with cystic spaces. It is called as *osteitis fibrosa cystica*. Bone becomes friable and leads to pathological fracture.

Features: Common in phalanges, jaw bones and skull bones. Pathological fracture can occur. Serum calcium is raised. Serum phosphorus is lowered. Serum alkaline phosphatase is increased. Serum PTH is raised. Thallium scan shows hyperfunctioning parathyroids. CT neck or MRI shows the nodule in parathyroid. X-ray of skull, hand, jaw shows *salt and pepper* lesion.

Osteoporosis

It is reduction in total skeletal bone mass leading to thinning of the cortical margins, less dense cancellous bone.

Causes: After menopause in women; In old age (elderly); Disused due to lesser activity.

Problems: These individuals are more prone for fracture by minor trauma, which is often unnoticed. Commonly observed fractures are Colles' fracture, fracture neck of femur, compression fracture of vertebrae, intertrochanteric fracture.

Scurvy

Deficiency of vitamin C causes defective endochondral ossification with more unossified cartilages leading to haemorrhages and swelling in the epiphyseal region. They present with haemorrhages, swelling, pathological fracture.

Osteogenesis Imperfecta (Brittle Bones)

It is a congenital (*recessive*) inherited disease with defective collagen synthesis leading to brittle bones which are prone for multiple fractures in multiple bones.

Features: Multiple fractures; Sclerosed ear (otosclerosis); Blue eyes (blue sclerotics); Ligament laxity; Blood chemistry normal; Bone histology normal. *Osteogenesis imperfecta tarda* occurs in puberty age group (late childhood) as *autosomal dominant* inheritance.

Achondroplasia

It is a familial congenital disease (autosomal dominant) where there is failure of normal ossification of long bones and skull bones.

Features: Long large head, normal trunk; short proximal part of the limb; lumbar lordosis; *trident hand* and wide pelvis side-by-side; mental impairment is *not* present (**Fig. 30.42**).



Fig. 30.42: Achondroplasia.

Diaphyseal Aclasia (Multiple Exostosis)

It is a growth disorder due to defective endochondral ossification with failure of remodeling of bone ends.

Features: It is commonly familial; Common in lower end of femur, upper end of tibia, humerus; Dwarfism

is common; Exostosis is peduncle with cartilage as cap with a bursa in between. It grows away from the joint surface.

Complications: Bursitis; Compression of neurovascular bundle and tendons; Restriction of joint movements; Turning into chondrosarcoma (5%).

Enchondromatosis (Ollier's Disease)

There is abnormal proliferation of cartilage cells of the growth plate into the metaphysis of long bones. It is commonly seen in bones of fingers, toes and other long bones. It also can turn into chondrosarcoma (5%).

In born defects of mucopolysaccharide metabolism causing dwarfism. They are—*Morquio Brailsford* disease (flat vertebrae, distorted hip, undue lax ligaments, keratin sulphate in urine); *Hurler's* disease (mental retardation, respiratory and cardiac complications, urine showing derman and heparin sulphate); *Hunter's* disease.

Marfan's Syndrome

Defect in elastin/collagen formation as autosomal dominant trait.

Features: Tall, undue lengthening of distal body segment, scoliosis, arachnodactyly (spider fingers), high arched palate, hernias, dislocation of ocular lens, aortic aneurysm. When it is associated with homocystinuria it is autosomal recessive.

Bone Tumours

It is either benign or malignant. Malignant can be either secondaries or primary. Secondaries are the commonest malignant bone tumour. Osteochondroma is the commonest benign bone tumour.

Benign Bone Tumours

Osteoma; Osteochondroma; Chondroma; Osteoblastoma.

Osteoma

It is a benign tumour arising from the surface of a long/flat/skull bone.

Types: (1) *Ivory osteoma* is hard compact. It usually occurs in skull bone like frontal bone/parietal bone.

It presents like localised bony hard swelling which is nontender, nonmobile, with free skin over the surface. It does not turn into malignancy. It can be left alone. Only if it is cosmetically troublesome it can be excised. But it should be differentiated from other swellings in the scalp like dermoid cysts or secondaries. Frontal bone osteoma may rarely extend into frontal sinus or orbital cavity and if it is so it needs surgical excision after doing CT scan. (2) *Cancellous osteoma* usually arises from spongy bone with a localised swelling. Features are similar like ivory osteoma (**Figs. 30.43 and 30.44**).



Fig. 30.43: Osteoma in the skull. Frontal bone is the common site.



Fig. 30.44: Patient with osteoma skull behind the ear.

Chondroma

It is a tumour arising from cartilage.

Types: (1) *Ecchondroma* grows outwards from the bone. It is common in flat bones like scapula/ilium or bones of hands and feet. In flat bones they often reach large size. Occasionally it may turn into malignancy as chondrosarcoma. (2) *Enchondroma* is more common in bones of hands and feet. The affected bone expands from within with thinning of the bone cortex. Pathological fracture can occur. If this type is not troublesome it can be left alone. (3) *Multiple chondromas* in major long bones is called as *dyschondroplasia/multiple chondromatosis* or *Ollier's disease*. It usually begins in childhood as enchondromatosis in the region of the growing epiphyseal cartilages of many bones. So there will be interference of the growth of the epiphyseal plates which causes shortening and deformity (**Fig. 30.45**).

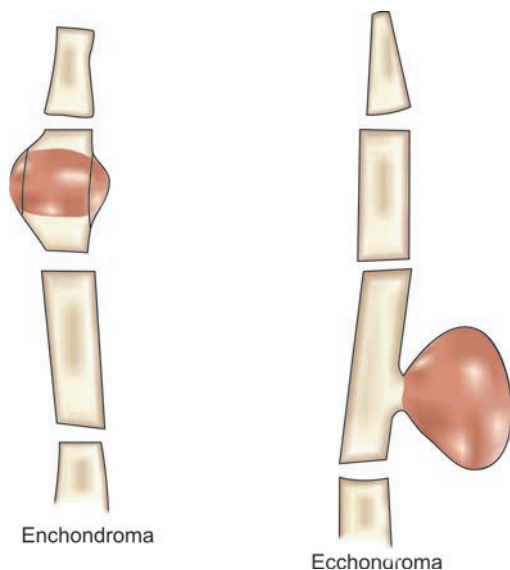


Fig. 30.45: Chondroma types. Ecchondroma grows from within. Enchondroma grows outwards.

Osteochondroma

It is commonest benign tumour of the bone. It begins in childhood from the growing epiphyseal cartilage plate. As the bone grows tumours is left behind and so appears like migrating towards the shaft of the bone. It grows outwards like a mushroom. Its stalk and proximal part is bony but distal part is cartilaginous

like a cap often with a bursa in between. Usually it is single but it can be multiple. Multiple osteochondromas involving several long bones is called as *diaphyseal, aclasis/multiple exostoses*. Osteochondroma should be excised only after completion of the development of the bone.

Complications: It often can compress neurovascular bundle. It presents with painless swelling. Only cartilaginous component turns into malignancy—chondrosarcoma. Osseous part will not turn into malignancy (**Fig. 30.46**).

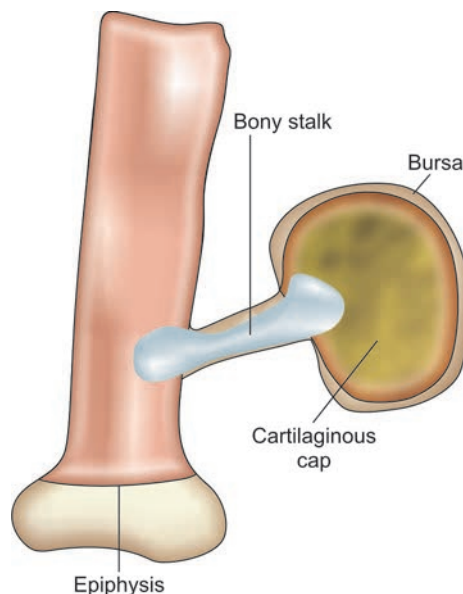


Fig. 30.46: Osteochondroma.

Osteoclastoma

Osteoclastoma often termed as *giant cell tumour*. It occurs in ends of long bones from epiphyses often extends into the joint cavity. It also occurs in jaw either mandible or maxilla. It can be benign/intermediate or malignant (10%). Malignant osteoclastoma spreads into lungs through blood. It forms an expanding tumour with localised swelling which is bony hard. It has got typical loculated appearance. Histologically it contains spindle cells (typical) with osteoclastoma giant cells. It can cause pathological fracture. X-ray/incision biopsy and CT/MRI are the needed investigations (**Figs 30.47 to 30.49**).

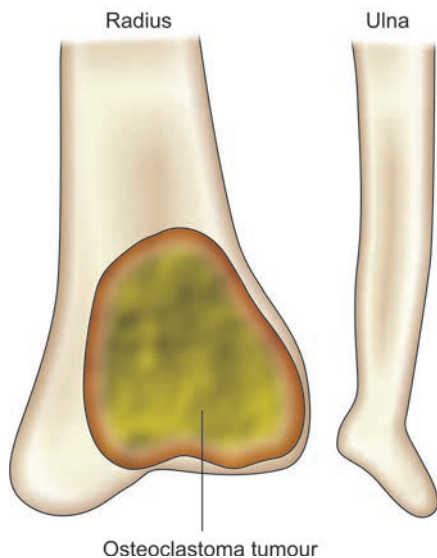


Fig. 30.47: Osteoclastoma.



Fig. 30.49: X-ray showing features of osteoclastoma of lower end of radius.



Fig. 30.48: Osteoclastoma of base of 1st metacarpal bone.

Malignant Bone Tumours

Secondaries is the *commonest* malignant tumour of the bone (**Figs 30.50 and 30.51**).

Primary Malignant Bone Tumours

Osteosarcoma; Chondrosarcoma; Fibrosarcoma of bone; Ewing's tumour; Multiple myeloma.



Fig. 30.50: Pathological fracture of humerus—primary from carcinoma of breast.

Common primaries causing secondaries in bone are

All sarcomas
 Carcinoma kidney (RCC)
 Carcinoma breast— 70% cases in females.
 Follicular carcinoma thyroid
 Carcinoma prostate
 Carcinoma lung

Common bones involved

Vertebral bodies
 Ribs and sternum
 Pelvis
 Upper end of femur and humerus

Types

Osteolytic— common
 Osteosclerotic—carcinoma prostate
 Combined osteosclerotic and osteolytic



Fig. 30.51: X-ray showing lytic secondaries in right femur.

Osteosarcoma: It is common primary malignant tumour of the bone. It is common in children/adolescence. Common sites are lower end of femur/upper end of tibia/upper end of humerus. It arises from metaphysis. It expands outwards extending into adjacent soft tissues. It spreads into lungs commonly through blood. It is very aggressive tumour. It causes extensive destruction of bone with rising of the periosteum and new bone formation. Pathological fracture is common. Localised pain, swelling which is warm, hard, and vascular are the features (**Figs 30.52A and B and 30.53**).

Investigations: X-ray shows tumour in the end of the long bone with cortical destruction, Codman's triangle, 'sunray' appearance, pathological fracture. Open incision biopsy, MRI of the lesion, CT chest to see secondaries.

Ewing's sarcoma: It is highly malignant endothelial sarcoma of bone arising from bone marrow. It begins in diaphysis. It is soft, vascular tumour arising



Figs 30.52A and B: Osteosarcoma of femur.

commonly from shafts of femur/tibia/humerus. It expands outwards with successive layer-by-layer formation of new bone. It commonly spreads through blood into lungs. It commonly occurs in children. It



Fig. 30.53: X-ray femur showing features of osteosarcoma.

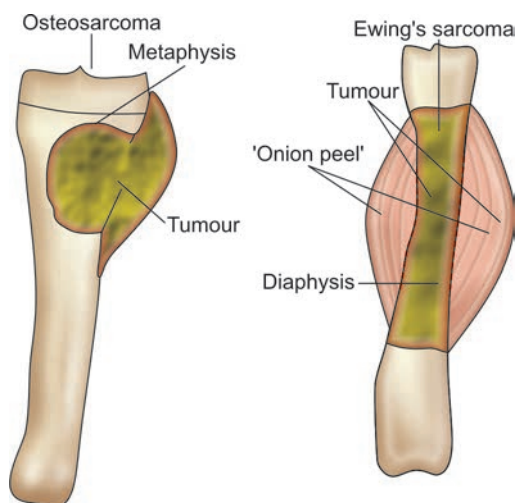


Fig. 30.54: Diagram showing osteosarcoma and Ewing's sarcoma.

presents as soft, vascular, firm, fusiform swelling in the shaft of long bones with warm skin over the tumour (**Fig. 30.54**). X-ray has typical 'onion-peel' appearance.

Multiple myeloma: It is malignant aggressive tumour arising from plasma cells of the bone marrow. It mainly involves spine, skull, flat bones and ends of long bones. Generalised pain and illness, anaemia, bone pain, pathological fracture, neurological deficits often with paraplegia are the features. X-ray shows multiple radiolucent areas in spine, pelvic bones, and skull bones. Blood smear shows 'cart-wheel' shaped plasma cells. Bence Jones proteins are positive in urine of the patient. Specific immunoglobulin will be elevated and is diagnostic. Bone marrow biopsy is essential. Radioisotope study of bone is useful.

Chondrosarcoma: It is common in flat bones like ilium, ribs. Swelling which is slowly progressive attaining enormous size with dull aching pain is the usual presentation. X-ray shows lytic lesion or often with calcifications. It spreads to lungs through blood.

Cysts of the Bone

Unicameral Bone Cyst (Solitary/Simple Bone Cyst)

It is seen in children and adolescents in long bones commonly (*proximal humerus*), occasionally in scaphoid or lunate. Cyst appears in the metaphysis in the *centre* containing *clear fluid*, appears as *uniform, vertically oval* lesion. Pain, discomfort, pathological fractures are the features. It should be differentiated from Brodie's abscess, aneurysmal bone cyst, hyperparathyroidism, secondaries.

Aneurysmal Bone Cyst

It occurs in adolescents as a *blown out* disease of bone of *unknown* aetiology. It is a *misnomer* as it is not related to artery. Cyst is *eccentric*, expands under periosteum often under soft tissues containing connective tissues, vascular spaces and *blood in fluid* status. Eccentric blown out look on X-ray is typical. Pathological fracture may occur.

EXAMINATION OF PATHOLOGICAL JOINT

Tuberculosis of the joint is common in children and adolescents. Rheumatoid arthritis is seen in adult female; osteoarthritis is seen in old age.

Sudden onset and rapidly progressive is a feature of acute arthritis; insidious onset, slowly progressive is seen in joint involved by tuberculosis, rheumatoid/osteoarthritis.

Sudden severe pain is seen in acute arthritis. Painless joint is a feature of Charcot's joint (Tabes dorsalis/syringomyelia) and Clutton's joint (congenital syphilis). Site of pain, referred pain (hip to knee), character (dull in chronic disease, severe throbbing in acute), *night cry* (in tuberculosis sleep protective muscle spasm disappears during night causing friction of diseased articular surfaces of the joint) should be asked. Whether pain increases by joint movement should be asked, *early morning pain* while getting up from bed is seen in osteoarthritis that gradually disappears after movements due to synovial fluid secretion into the joint cavity.

Fever, its severity, night fever is significant.

Joint locking due to loose bodies is common in knee joint.

Deformity, its duration, occurrence should be asked.

Past history of tuberculosis, syphilis, trauma; family history of haemophilia, gout, tuberculosis should be asked.

Examination

Inspection

Normal and affected joints should be inspected. Joints proximal and distal should be inspected. Inspection should be done from all sides.

Swelling is inspected from all sides of joint. It can be generalised or localised. Fullness may be due to synovial thickening of the joint also.

Position of the joint: Joint when filled with fluid, takes the position of ease. It is plantar flexion and inversion for ankle joint; slight flexion for knee; slight flexion, external rotation and abduction for hip; slight flexion for wrist; flexion and slight pronation for elbow; slight flexion, adduction and internal rotation for shoulder.

Deformity and muscle wasting (quadriceps in knee disease; gluteal muscles in hip disease).

Skin over the joint for features of acute inflammation or scar, sinuses, discharge, ulcer, etc.

Palpation

Temperature over the joint is checked using back of the hand, first on sound joint then on diseased joint.

Tenderness on joint line, ligament, and bone should be checked.

Swelling: Joint effusion takes the form of the joint. Fluctuation can be elicited in the joint. Bursa when enlarged forms a localised swelling where joint is normal. Communicating swelling will be localised but with a joint effusion underneath. Synovial thickening is confirmed by its elastic/boggy/spongy nature. It is seen in tuberculosis, rheumatoid arthritis.

Movements: Limitation of all movements is a feature of acute arthritis. Both active (by the patient) and passive (by the examiner) movements should be checked. Normal side should be examined first. When movements are checked, confirm whether it is painful or not; has to be confirmed; if so which movement is painful, angle at which pain appears and at which angle it disappears (if at all); presence of any muscle spasm (suggests active disease); presence or absence of restriction of movements, if present whether all movements or only certain movements are restricted; presence of any crepitus during joint movements (osteoarthritis) should be seen.

Measurements: Length of each segment of the limb and circumference of the limb should be checked. It should be compared to opposite side.

Crepitus of joint can be auscultated also.

Regional lymph nodes should be examined. In upper limb joints axillary and neck nodes; in knee and ankle inguinal nodes; in hip joint external iliac nodes are affected.

Other joints in the limb, opposite side and other parts of the body also should be examined.

Systemic examinations like of respiratory system (tuberculosis), cardiac system (rheumatic fever), neurological (CNS) system (in Charcot's joint with tabes dorsalis or syringomyelia) should be done.

Investigations

Blood: Total count is increased in acute infection. ESR is raised in tuberculosis. Bleeding time, clotting time is altered in haemophilia.

X-ray of joint to see articular surfaces, widening of space, loose bodies.

Joint fluid aspiration: For culture, cytology, AFB. Synovial biopsy is very useful.

Arthroscopy is very useful and is diagnostic. It is not done in acute suppurative arthritis.

Gallium or technetium isotope scan is used in inflammation.

MRI of joint is very useful investigation.

Other tests: Blood uric acid, VDRL/Kahn test for syphilis; Rose Waaler test for rheumatoid arthritis; Mantoux test for tuberculosis; FNAC of lymph node.

Acute Pyogenic Arthritis

It is acute infection of a joint caused by *Staphylococcus*, *Streptococcus*, *Pneumococcus* organisms either through haematogenous spread from distant focus or trauma. Exudates may be serous, serofibrinous or purulent depending on the severity of infection. Pus formation in the joint leads to destruction of articular cartilage; it may track through the soft tissues and skin causing abscess and sinus. Often severe virulent infection causes septicaemia and if not treated properly death may ensue.

Clinical features: Pain and swelling in the joint; restricted joint movement, diffuse tenderness, warmth, redness, and fullness over the joint; joint is in maximum ease position; spasm of the muscles adjacent to the joint. Active movement are absent and passive movement are painful. Pneumococcal arthritis occurs in children due to spread from ear, throat, lungs, common in knee joint, often painless purulent joint is the presentation, aspirated pus is creamy green. If there is destruction of joint surfaces, it leads to bony ankylosis (fusion of joint). Arthritis can occur in infants, in patients with gonococcal infection and also syphilis. Gonococcal arthritis is acute, polyarticular, occurs as blood borne infection after 3 weeks after primary infection (or once urethral discharge has stopped).

Reiter's disease—Acute polyarthritis (with sacroiliac joint), nonspecific urethritis, conjunctivitis.

Chronic Pyogenic Arthritis

It is due to chronic recurrent infection of the joint by gram positive or other bacteria. It causes deformity, disability and eventually bony ankylosis of the joint.

Rheumatoid Arthritis

It is an autoimmune connective tissue disorder involving many systems including skeletal system. In joints, it causes inflammation of the synovial membrane which gets thickened, oedematous, and vascular. Eventually articular cartilage also gets involved in inflammatory process leading to formation of granulation tissue called as *pannus*. *Pannus* eventually extends to capsule, periarticular tissue and deeper bone surfaces. Finally joint ankylosis, muscle atrophy occurs.

Clinical feature: Common in women; It has got different phases with remissions and exacerbations. Small joints of hand and feet are involved first. Later proximal joints are involved. Morning stiffness and pain are typical. Restricted painful joint movements with effusion and swelling of the joint occur. Flexion deformity of fingers and toes occurs often with *buttonhole* or *swan neck* deformity. In severe cases patient may be permanently crippled. Symmetrical joint involvement is seen. Patient may present with carpal tunnel syndrome, tennis elbow, plantar fasciitis, rheumatoid subcutaneous nodules, vasculitis with ischaemia or gangrene of fingers or toes, pleural effusion, pericardial effusion, muscle wasting.

Investigations: X-ray shows narrowed joint space with subchondral cystic areas; High ESR; IGM Rheumatoid factor is positive; Synovial fluid shows high cell count ($> 10,000$) and high protein (> 5 gm%); Synovial biopsy is diagnostic; Arthroscopy is very useful.

Osteoarthritis

It is a degenerative disease of the joint. There is degeneration of articular cartilage due to wear and tear. There is low grade or no inflammatory process. There is fragmentation and fibrillation. Cartilage when gets thinned out, bone surface is exposed. Reactive hypertrophy is observed in peripheral margins of the bone surfaces which forms osteophytes. Ankylosis of joint is not common. As there is not much of inflammatory reaction it is better called as osteoarthritis not as osteoarthritis.

Types: *Primary osteoarthritis* occurs in weight bearing joints like knee, hip and spinal joints. It is common in old age, women, and obese individuals. *Secondary osteoarthritis* is due to other diseases in the joint like avascular necrosis, trauma and so on. It is due to mechanical incongruity of the articular surfaces. *Common joint involved is knee, then hip.*

Clinical features: Pain, stiffness, difficulty in squatting; muscle wasting, position of ease; restricted movements, disability; joint effusion and swelling; crepitations over the joint.

X-ray reveals narrowed joint space with subchondral sclerosis and osteophytes over the margins of the articular surfaces. Osteoarthritis is a morbid condition.

Tuberculous Arthritis

It is usually secondary to primary pulmonary or urogenital tuberculosis. Lower limb joints are commonly involved (hip/knee). It is common in children; *night cry* due to absence of protective muscular spasm during night sleep; restricted all movements due to protective muscle spasm; muscle wasting; cold abscess/sinus formation. Disease can be osseous (hip) or synovial (knee) or combined (spine). Synovial thickening is common. X-ray, fluid aspiration, ESR, Mantoux test, synovial biopsy are needed investigations. Fibrous (common)/bony ankylosis (if joint has secondary infection); disability, shortening of limb, cold abscess/sinus formation occurs later.

Gouty Arthritis

There is high serum uric acid due to more production or poor excretion. First metatarsophalangeal joint (great toe) is involved commonly, later other toes, fingers, wrist and ankle. Urate crystals deposited causes acute painful arthritis. Later osteoarthritis of joint sets in. X-ray shows *tophi* in the joints and different parts of the body.

Clutton's Joint

It is seen in congenital syphilis with interstitial keratitis.

It is painless symmetrical synovitis commonly of both knee joints with wasting of thigh muscles.

Osteochondritis

Crushing osteochondritis

Avascular necrosis of epiphysis of unknown cause. Pain, spasm, irritability, deformity are the features.

1. Hip (hemoral head)—Perthes' disease.
2. Thoracic spine—Schürmann's disease.
3. Lunate bone—Kienbock's disease.
4. Navicular bone—Kohler's disease.
5. 2nd metatarsal—Freiberg's disease.

Splitting osteochondritis dessicans

Trauma causing avascular segment—splitting—loose bodies.

Common in adolescents.

1. Knee joint is commonest site. Medial femoral condyle is the site. Pain, tenderness, locking due to loose bodies is the features.
2. Elbow is second common site. Capitulum of radial head is affected.
3. Ankle is rare site. Upper surface of talus is affected.

Traction osteochondritis

Occurs at site where tendon is attached to epiphysis/apophysis.

1. At the attachment of ligamentum patella to tibial tubercle—Osgood Schlatter's disease.
2. At the attachment of tendo Achilles to calcaneum—Sever's disease.
3. At the lower part of patella attaching to ligamentum patella—Johansson-Larsen disease.

Ankylosis

It is stiffness of joints. It can be intraarticular, *true ankylosis* or extraarticular, *false ankylosis*. It can be painless/painful ankylosis.

True ankylosis: Types: (1) *Fibrous*—It is due to fibrosis of two articular surfaces; it is seen in—acute or tuberculous or gonococcal arthritis. (2) *Bony*—Severe destruction of articular cartilages causes formation of bony trabeculae between the two articular ends.

False ankylosis: It is due to fibrosis of extraarticular tissues near the joint causing limitation of joint movement. It can be due to burns contracture (skin); muscle contracture (Quadriceps/Volkmann's); fascial (Dupuytren's); contracture of capsule and ligaments; myositis ossificans traumatica.

EXAMINATIONS IN INDIVIDUAL JOINT PATHOLOGY

Examination of Pathological Shoulder Joint

Pain and stiffness of the shoulder is the main complaint. Pain may also be due to referred pain from diaphragm, neck, chest or abdomen.

Inspection

Position is flexed, medially rotated with arm on side of chest in diseases. In different arthritis deltoid *wasting* causes *flattening*. Joint is prominent and *rounded* in joint *effusion* (extends on anterior and posterior margins of deltoid and along long head of biceps) or subdeltoid/*subacromial bursitis* (a cystic swelling extends only beneath the deltoid with normally palpable humeral head) (**Fig. 30.55**).

Opposite shoulder, arms, elbows, axilla, neck should be inspected.



Fig. 30.55: Shoulder should be inspected from all around.

Palpation

It is done with patient *sitting* non a stool, keeping his arm beside the chest wall (**Fig. 30.56**).

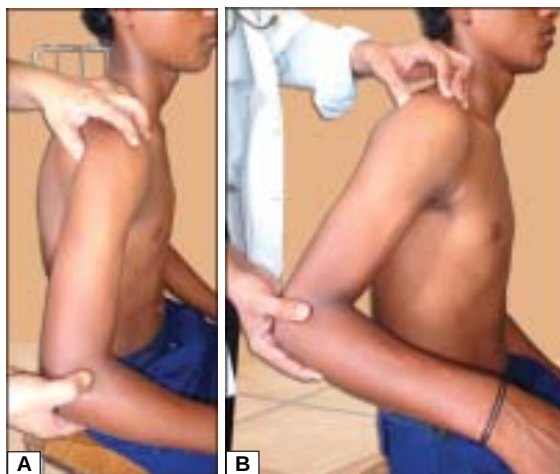
Tenderness is felt below the acromial process in supraspinatus tendinitis or only during adduction in painful arc syndrome; tenderness over coracoid process are felt in certain other joint pathologies. Crepitus over joint suggests osteoarthritis. Tip of coracoid (in front), tip of acromion (above and behind), greater tuberosity should be felt.

Swelling should be palpated and confirmed by palpating across axilla.



Fig. 30.56: Shoulder is palpated in sitting position with arm on the side of the chest wall.

Codman's method of palpation: Left hand is used for right shoulder and vice versa. Using thumb placed below the spine of scapula, posterior part of the shoulder is palpated. Index finger is placed anterior to acromion to palpate the superior and part of anterior part of the joint. Other three fingers are kept over the clavicle to hold. Patient's flexed elbow is held with right hand and flexion and extension movements are done while palpating the shoulder (**Figs 30.57A and B**).



Figs 30.57A and B: Codman's method of shoulder joint palpation.

Movements: Movements should be examined both from front and behind, and compared to opposite side. Plane of body of scapula is not coronal but 30° to this plane. Flexion of arm is forward and medially; extension is backward and laterally. In abduction initial 30° movements is by shoulder joint alone; further up to 120° , for every 15° movement 10° is from glenohumeral and 5° from scapula. Scapula should be held firmly to find out the range of scapular movement.

Abduction is 180° ; flexion is 90° ; extension is 45° ; rotations medial/lateral, each is 45° ; all movements together form circumduction.

Abduction and external rotations are most commonly affected. Beginning 30° of abduction is by action of supraspinatus and it is difficult if this tendon is ruptured completely. If arm is assisted passively for this movement patient can continue the remaining abduction actively as beyond 30° , abduction is done by deltoid (**Figs 30.58 to 30.64**).

Mid range abduction 60° - 120° is affected and painful in chronic supraspinatus tendinitis as degenerated tendon impinges between under surface of acromion and greater tuberosity of humerus—*painful arc syndrome*. Beginning and end of abduction is painless. It is sometimes also seen in subdeltoid bursitis, incomplete rupture of the supraspinatus tendon, crack fracture of greater tuberosity of humerus.

Entire range of abduction is painful in acute arthritis and acute supraspinatus tendinitis (localised degeneration of supraspinatus tendon in young adult often with



Fig. 30.59: Abduction of shoulder joint.



Fig. 30.58: Passive movement of the shoulder joint is done by fixing the scapula with one hand.



Fig. 30.60: Adduction of shoulder joint.



Fig. 30.61: Flexion of shoulder joint.



Fig. 30.62: Extension of shoulder joint.

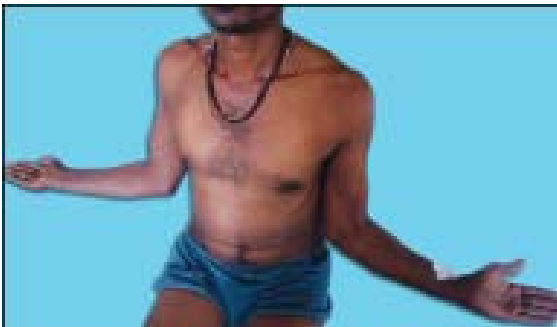


Fig. 30.63: External rotation of shoulder joint.



Fig. 30.64: Internal rotation of shoulder joint.

calcification and acute oedema causing painful abduction which disappears once calcified part bursts into subdeltoid bursa).

Sharp pain appears only when the arm is raised above the shoulder level in case of arthritis of acromioclavicular joint.

Examination of acromioclavicular and sternoclavicular joints is important.

Tuberculosis of Shoulder Joint

Tuberculosis of shoulder joint which causes restriction of shoulder joint movement is called as '*caries sicca*'. Often there will be cold abscess or sinuses called as *florid* type.

Rotator Cuff

Rotator cuff is a tendinous cuff formed by subscapularis in front, supraspinatus above, infraspinatus and teres minor behind which fuses with shoulder joint capsule. Its injury also restricts the abduction.

Frozen Shoulder

It is common in elderly women. There is adhesion between synovial membrane and joint capsule. *I stage*: There is pain more at night and with gradual progressive stiffness with restriction of abduction and external rotation; *II stage*: Pain gradually subsides but stiffness persists; *III stage*: Disappearance of stiffness and

regaining normal shoulder movements. Each phase lasts 6-8 months with total duration of 2 years.

Examination of Pathological Elbow Joint

Pain, stiffness, deformity are the main problems.

Inspect the carrying angle, position (position of ease—semiflexed), swelling (effusion with fullness over cubital fossa, olecranon bursa, bicipitoradial bursitis), muscle wasting.

Joint effusion first obliterates the concavity on each sides of olecranon (as synovial cavity is nearest at this region and posterior ligament is thin and lax), eventually swelling is noticed over posterolateral part of elbow over radiohumeral joint eliciting cross fluctuation.

Supratrochlear lymph node should be examined for enlargement—is checked in front of medial intermuscular septum 1-1.5 cm above the medial epicondyle with elbow in flexed position.

Tennis Elbow

Tennis elbow is lateral epicondylitis involving common extensor origin. Presents with pain, and tenderness, that aggravates on dorsiflexion of wrist. Patient develops pain when he extends his clenched fist against resistance—*Cozen's test*. Patient complains of severe pain at common extensor origin when his pronated forearm is passively flexed—*Mill's manoeuvre* (Figs 30.65 and 30.66) (Refer Chapter 9, page 223).

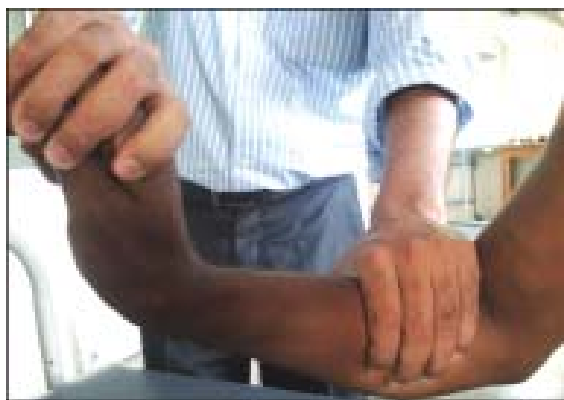


Fig. 30.65: Cozen's test in tennis elbow.



Fig. 30.66: Mills manoeuvre used in tennis elbow.

Golfer's Elbow

Golfer's elbow (*Basebaker's*) is similar and rare on medial epicondyle at common flexor origin. *Olecranon bursitis* is called as **student's/Miner's elbow**.

Cubitus Valgus

It is increase in carrying angle more than normal (male—10°; female—15°) with extended forearm showing excessive abduction in relation to arm. *Causes* are previous fracture of lower end of humerus or capitulum with malunion, injury or infection of lateral aspect interfering lateral epiphyseal growth. It causes angulation of the *ulnar nerve* medially with repeated friction of nerve leading into ulnar nerve palsy.

Cubitus Varus

It is opposite of valgus showing decreased or reversed carrying angle in extended elbow. Causes are supracondylar fracture of humerus with malunion; epiphyseal growth interference on medial side. It is less troublesome than valgus.

Examination of Pathological Wrist and Joints of Hand

Position of ease is flexion with ulnar deviation. Congenital anomalies are common. Compound palmar ganglion (ventral), ganglion (dorsal) are common. *Joint effusion* is not common. When it is present swelling appears, both in front and behind the wrist and is palpable which is cross fluctuant. In arthritis tenderness is elicited along the joint line; in tenosynovitis tenderness is elicited along the tendon sheath.

All movements are painful and limited in arthritis. (Refer Chapters 9 and 27).

de Quervain's Stenosing Tenosynovitis

It is degenerative thickening, inflammation and later stenosis of abductor pollicis longus and extensor pollicis brevis in middle aged women near radial styloid process in anatomical snuff box under common synovial sheath. Localised tender swelling may be present with painful thumb extension against resistance. Passively adducted wrist or ulnar deviated thumb causes pain. With the flexed thumb in the palm covered by fingers firmly, hand is passively deviated medially to cause pain at radial styloid process which shoots below to thumb and above to elbow—*Finkelstein's test*.

Phalen's sign (wrist flexion test): It is done for carpal tunnel syndrome. Flexing the wrist for 1-2 minutes causes the exacerbation of symptoms with paraesthesia and symptoms disappear once wrist is straightened. *Reverse Phalen's sign* is similar but wrist placed in extension position (For details about carpal tunnel syndrome refer chapter 8).

Examination of Pathological Hip Joint

Pain often radiating into knee is common; night cry is also common. Limp either painful, waddling (bilateral congenital dislocation), lurching on towards affected side (Trendelenburg gait) should be asked. Congenital dislocation of hip occurs at birth; Perthes' disease from 5-10 years; slipped femoral epiphysis from 10-15 years; tuberculosis of hip at any age group can occur.

Inspection

Entire hip, pelvis, spine should be observed in standing position. Inspect the attitude, muscle wasting of glutei and adductors, swelling (cold abscess in femoral triangle or gluteal region), scar or sinuses, other joints.

Attitude

Slight flexion of diseased hip is common. Patient bears his weight on normal side.

Trendelenburg test: Patient when stands on normal limb, buttock/pelvis on diseased side rise. Patient when stands on diseased side opposite normal side pelvis sink as diseased side abductors' pull is defective. This means test is positive. It is seen in weak abductors (polio, motor neuron disease); unstable fulcrum to act (dislocation of hip); loss of liver system (fracture neck of femur, coxa vara, Perthes'); arthritis of hip.

Fixed abduction or adduction deformity: Limb when fixed in abduction there will be scoliosis of lumbar spine with convexity towards affected side with downward tilt of pelvis and apparent lengthening of the limb. Limb when fixed in adduction there is scoliosis of lumbar spine with convexity towards normal side with upward tilt of pelvis and apparent shortening of the limb. Line joining two anterior superior iliac spines is normally horizontal and right angle to midline. This line will be at lower level in affected side in abduction deformity; higher in affected side in adduction deformity. *In abduction deformity*—the affected leg is held just above the foot, and is abducted till interspinous line becomes horizontal and angle at which line becomes horizontal is *actual angle of fixed abduction deformity*. Similarly angle of fixed adduction deformity is checked by adducting the limb until line becomes horizontal (**Fig. 30.67**).



Fig. 30.67: Interspinous line should be made horizontal by adducting or abducting the limb depending on the adduction/abduction deformity to make the interspinous line horizontal.



Figs 30.68A and B: Fixed flexion deformity of hip is compensated by lumbar lordosis. Angle of fixed flexion deformity is checked by flexing the normal knee and hip over the abdomen.

Fixed flexion deformity: Limb when fixed in flexion there will be lumbar spine lordosis. Patient extends the limb by bending lumbar spine forward which can be confirmed by ability to place hand under the lumbar spine when patient lies in the bed supine with limbs straight. Now normal thigh and flexed knee of the patient is flexed towards abdomen until lumbar lordosis disappears which is confirmed by inability to pass hand behind the lumbar spine. By this diseased limb automatically will flex and angle from bed to flexed limb is considered as *angle of fixed flexion deformity* (*Hugh Owen Thomas test*) (**Figs 30.68A and B**).

Fixed medial or lateral rotation deformity: Normally lower limb is in slight lateral rotation. It is clinically assessed by looking at the anterior surface of the patella or toes with foot at right angle to leg. If it is pointing towards the ceiling it means there is medial rotation.

By flexing both the hips and knees limb shortening can be assessed by observing the level of knees.

Congenital dislocation of hip: There is marked lordosis, anterior protrusion of abdomen, buttock protruding behind, and broadened space below the perineum in bilateral disease. Waddling gait is observed (bilateral case). Disease is common in girls. It is

common in Europe and Japan; rare in Negroes. If it is unilateral, grooves between thigh and labia are asymmetrical with additional skin crease on the medial side of the thigh will be present. Abduction and rotations are limited; flexion and extension are normal; adduction is in excess. *Trendelenburg gait* with pelvis lurching towards diseased side is seen (unilateral case). Stability of hip joint is lost here (telescopic test, Ortolani's test, Barlow's test are positive). Movements are painless. *Vascular sign of Narath* is present. In X-ray, *Perkin's line* (upper femoral epiphysis is above the horizontal line of triradiate cartilage and outer to vertical line along the outer margin of acetabulum), *acetabular angle* (widened from normal 22° to 45°), *Von Rosen femoral lines* (line of femoral shaft extending upward which normally touches acetabular roof will be displaced above into the pelvis) are useful.

Slipped femoral epiphysis (adolescent coxa vara):

There is increased external rotation with adduction; but limitation of internal rotation and abduction. It is common in boys between 10-15 years. Trendelenburg sign is positive. In X-ray, line from upper part of neck passes above the head instead of passing on superior margin of head as in normal (*Trethowan's sign*). Metaphysis lies lateral to posterior acetabular margin instead of normal medial location (*Capener's sign*). Epiphysis is displaced backwards and downwards; neck shaft angle is reduced (normal is in child 150° , in adult 127°). Lesser trochanter is prominent.

Tuberculosis of hip: *Stage I*—There is joint effusion with position of ease being flexion, abduction, external rotation with downward tilt of pelvis and *apparent lengthening* of the limb. *Stage II*—Involvement of articular cartilage is seen with spasm of adductors and flexors causing flexion, adduction, and internal rotation and upward tilting of pelvis along with *apparent shortening* of the limb. *Stage III*: There is erosion of upper part of acetabulum with dislocation of femoral head with spasm of adductors causing *wandering acetabulum* with *true/real shortening* of the limb with similar attitude like stage II. *Note*: Abduction is the first movement to be restricted in tuberculosis of hip. *Babcock's triangle* on the lower margin of neck close to head is affected first. Pain radiating to knee, night cry, and limp are typical. It is 2nd common site of joint tuberculosis after spine.

In **Perthes' disease** abduction and internal rotation are restricted. Other movements are normal. There is avascular necrosis of femoral head. It is common in boys of 5-10 years age. Muscular wasting, positive Trendelenburg sign; deformity, 10% are bilateral.

In **osteoarthritis of hip pain**, stiffness, limp, wasting of glutei, limitations of all movements with X-ray showing osteophytes is the presentation. Causes are—trauma, Perthes' disease, tuberculosis, slipped epiphysis, etc. (**Fig. 30.69**).



Fig. 30.69: X-ray showing features of Perthes' disease—flattening of femoral head.

Palpation

Tenderness is elicited over joint line below the midinguinal point. It is also elicited by applying pressure inwards over the two greater trochanters.

Greater trochanter is easily palpable for tenderness, broadening, and upward displacement.

Swelling: Joint effusion is felt with fluctuation just below the midinguinal point. Cold abscess due to hip joint tuberculosis can occur at—in front and medial to greater trochanter; medial to femoral vessels; in the gluteal region; in the pelvis if there is wandering acetabulum. Abscess in the pelvis may project at ischiorectal fossa to form sinus or fistula.

Femoral artery may not be felt in dislocation of hip—vascular sign of *Narath*.

Other joints should also be examined properly—knee, sacroiliac, spine, opposite side.

Movements in Hip Joints

Flexion: With the knee extended (tensed hamstrings) flexion of hip is 90°. With the knee flexed flexion

of hip is up to apposition of thigh to abdomen—120°. Pelvis is grasped firmly to prevent pelvic tilt and hip is flexed to assess the range. In fixed flexion deformity, initial Thomas test is done to find out the angle of fixed flexion. Any flexion beyond this angle is the existing free flexion of the diseased side.

Extension: It is checked in prone position with knee flexed 90°. Normal extension is 15°. Extension is absent in fixed flexion deformity (**Fig. 30.70**).

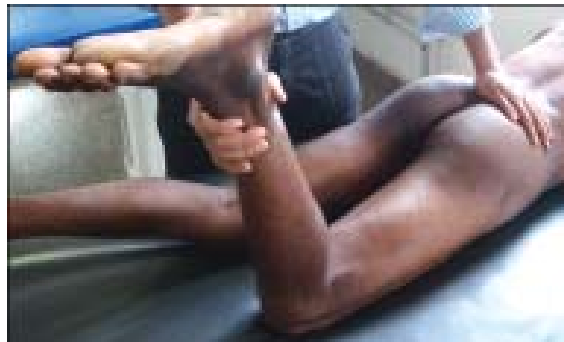


Fig. 30.70: Hip extension is checked in prone position with knee flexed (done when there is no fixed flexion deformity).

Abduction: Normal abduction is 40°. It is checked in supine position. Initially iliac crest on affected side is held to steady the pelvis. Thumb and middle fingers of the hand are used to keep over both anterior superior iliac spines (to identify the pelvis movements). In another method, patient in supine position both hips and knees are flexed, bringing soles close together, knees are then bent outwards to get the range of abduction (**Fig. 30.71**).



Fig. 30.71: Abduction of hip with flexed knees.

Adduction: Normal range is 30° . Normally limb can be crossed over the middle third of opposite thigh. After steadying the pelvis, limb is crossed over the opposite thigh (**Fig. 30.72**).



Fig. 30.72: Checking abduction and adduction of hip with extended knee.

Rotation: External rotation is 45° . Internal rotation is 30° . With patient in supine position, both knee and hip in extended position, rotation is checked by moving flat of the hand over thigh and degree of rotation is checked by observing foot. It can be checked in supine position by flexing knee and hip at right angles; or in prone position by flexing the knee at right angle (**Figs 30.73 to 30.75**).

Tests for Stability of Hip Joint

Telescopic test: Pelvis is fixed with one hand touching the greater trochanter. Hip is flexed to 90° , and with the other hand knee is grasped to push thigh posteriorly in its axis to feel the posterior movement of greater trochanter in unstable hip. This positivity is felt in dislocations of hip and Charcot's hip (**Fig. 30.76**).

Ortolani's test: With both the hips and knees in flexed position, thigh of the diseased side is gradually abducted to develop a click of entrance of femoral head to acetabulum and click of exit is felt while releasing the compression.



Fig. 30.73: Checking the rotation of hip with hip and knee in extended position.



Figs 30.74A and B: Checking the rotation of hip in supine position with hip and knee flexed at right angle. This method is used in fixed flexion deformity.



Fig. 30.75: Checking the rotation of hip in prone position with knee flexed at right angle, this method cannot be used when there is fixed flexion deformity.

Barlow's test: Both hips and knees are flexed. Folded both lower limbs are held with both hands, placing fingers over the greater trochanters. Limbs are adducted



Fig. 30.76: Telescopic test for stability of hip.

completely to feel dislocation of femoral head from the posterior margin of the acetabulum with a clear click. Limbs are gradually now abducted to feel reduction of femoral head into the acetabulum with a click again.

Measurements

Length: Interspinous line should be made horizontal before taking the limb measurement. It is done by abducting or adducting the limb that depends on abduction or adduction deformity which patient is having. Sound normal limb while measuring for comparison should also be abducted or adducted to the same level/angle as diseased limb. Limb is measured from anterior superior iliac spine to medial malleolus. Thigh is measured from anterior superior iliac spine to the inner aspect of the joint line of knee (**Fig. 30.77**).



Fig. 30.77: Thigh is measured from anterior superior iliac spine to the inner aspect of the joint line of knee.

Circumference of the thigh is measured at a specific distance from anterior superior iliac spine and opposite side is also measured at same level. It is done in muscular wasting.

Shortening of greater trochanter is seen in dislocation of hip, slipped femoral epiphysis, Perthes' disease, suppurative arthritis. It is confirmed by drawing Bryant's triangle, Nelaton's line.

Other Examinations

External iliac lymph nodes are palpated in hip joint pathology.

Rectal examination should be done to see pelvic abscess in tuberculosis of hip.

Systemic examinations like of respiratory system, abdomen should be done.

X-ray: *Shenton's line* extends from upper curved part of obturator foramen to the lower border of neck of femur as a continuous arched line. It is altered in pathological dislocation of hip, Perthes' disease and tuberculosis of hip. *Perkin's line*, *acetabular angle*, *Von Rosen* femoral lines (see above) are useful in congenital dislocation of hip.

Examination in Pathological Knee Joint

History of trauma, pain, stiffness, limp, locking, and click should be asked.

Inspect knee joint from all sides, popliteal fossa, opposite knee, other joints, spine, gait, muscle wasting (in thigh it is more).

Attitude: Position of ease is moderate flexion occurs in initial effusion phase. Later in destruction phase—triple displacement occurs—flexion, posterior subluxation and lateral rotation of tibia with spasm of hamstrings. Genu valgum, varum, recurvatum should be checked. Position of patella is a good indicator of the deformity.

Swelling: Joint effusion obliterates all hollowness around the patella causing horse shoe swelling. Bilateral knee joint effusion is seen in Clutton's syphilitic joint, haemophilia, bilateral trauma. Extra-articular swellings like prepatellar bursitis (*housemaid's knee*—between skin and patella), infrapatellar bursitis (*Clergyman's knee*—between skin and lower

part of patellar ligament), *Morrant Baker's cyst* (posterior herniation of synovial membrane through oblique popliteal ligament in midline at lower part of the popliteal fossa, below the joint line which is more obvious on extension and disappears on flexion, is due to underlying osteoarthritis with effusion), semimembranosus bursa. *Cyst of the lateral semilunar cartilage* is on only one side (lateral) side of ligamentum patellae.

Palpation

Fluctuation: With one hand suprapatellar pouch is pushed, with the fingers and thumb of other hand fluid movement is felt on either side of patella (**Fig. 30.78**).

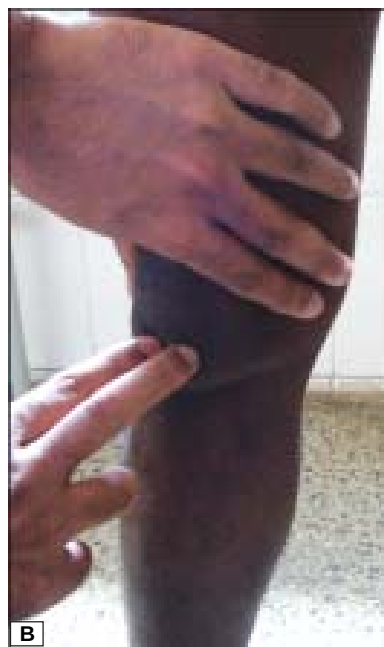


Fig. 30.78: Fluctuation in knee joint effusion is checked using fingers of both hands in an extended knee.

Patellar tap: It is done in patient in lying down position, with *fully extended knee*. With one hand suprapatellar pouch is pushed downwards to empty its fluid into joint proper, with the index finger of other hand patella is pushed behind sharply with jerky movements towards femoral condyles to feel 'strike' of patella over femoral condyles. Small quantity of fluid can be elicited by 'patellar tap' done in standing position or after applying pressure over the obliterated hollow on either sides of the patellar ligament in standing and releasing the pressure to appreciate slow refilling of the hollow (**Figs 30.79A and B**).

Synovial thickening is a boggy swelling on either side of patella which can be fluctuant but without patellar tap. It is commonly seen in tuberculosis.

Popliteal fossa should be examined in prone position with knee joint flexed. Lymph nodes, swellings,



Figs 30.79A and B: Classical method of eliciting the patellar tap in lying down position in extended knee. Small effusion can be checked as 'patellar tap in standing'.

pulsations, compressibility, reducibility, transillumination should be checked (**Fig. 30.80**).

Bony components should be palpated for tenderness, irregularity. Patellofemoral and tibiofemoral components of knee should be examined.

Intra-articular painful click is significant and is commonly seen in osteoarthritis.



Fig. 30.80: Popliteal fossa should be examined in knee joint pathology.

Movements: Flexion, extension, abduction, adduction and slight rotation in a flexed knee.

Measurements: Wasting of quadriceps, length of thigh, intermalleolar distance in genu valgum should be measured. Line from anterior superior iliac spine if extended downwards through middle of patella reaches the medial malleolus; this cannot be achieved in genu varum.

Popliteal, inguinal lymph nodes are to be examined. Abdomen, respiratory systems are examined.

Tuberculosis of knee joint (tumour Alba/white knee): It is secondary synovial type causing synovial thickening, *pannus formation*; later only becomes osteoarticular. Pain, night cry, fever, gait changes, loss of weight, joint effusion, thickening of synovium, thigh muscle wasting, *triple deformity*—posterior subluxation and lateral rotation of tibia—are typical features. Arthroscopy, synovial biopsy is diagnostic.

Knock knee (Genu valgum): It is abnormal abduction of the knee. It may be due to rickets, metabolic causes, epiphyseal diseases. It is assessed in standing position by measuring distance between medial malleoli. Pathology on the lateral aspect of tibial or femoral condyles causes abnormal increase in abduction and genu valgum. If it is femoral component deformity disappears on flexion but not if it is due to tibial component. Later osteoarthritis is the complication.

Bow leg (Genu varum): It is abnormal adduction due to all causes mentioned for knock knee wherein there is pathology on the medial aspect of the articular surfaces of tibial or femoral condyles. *Blount's disease* is an epiphyseal dysplasia seen in West Indies involving posteromedial aspect of proximal tibial epiphysis resulting in bow leg. Physiological bow leg is common in newborn which disappears spontaneously in 3 years.

Calcaneal Spur

It is prominent projection of calcaneal bone on its inferior aspect causing friction and pain in heel. Pain and tenderness is typical. Often fasciitis can occur underneath (**Fig. 30.81**).

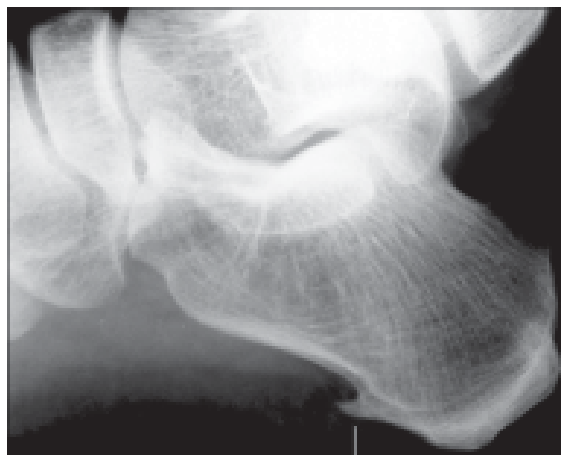


Fig. 30.81: Calcaneal spur. It is one of the causes for heel pain.

EXAMINATIONS IN SPINE INJURIES AND DISEASES

Spinal Injuries

History of accident/injury/its type/severity should be noted. Fall from height, severe violence can cause fracture dislocation of cervical spine. Seat belt injury can injure lumbar vertebra. History of pain in the spine, neck girdle, legs should be asked. History of numbness, paraesthesia in limbs is important. Dislocation is not common in thoracic/lumbar vertebra. Indirect violence causes injury commonly at C₆ and L₁ vertebrae. Paraplegia/quadruplegia may occur from spine injury.

Examination

Attitude

Injury above C₅ causes immediate death due to paralysis of phrenic nerve.

Injury at C₅ causes completely immobile paralysed body.

Injury at C₆ causes paralysed body with arm abducted (deltoid and supraspinatus); externally rotated (infraspinatus and teres minor); forearm flexed and supinated (biceps) (**Fig. 30.82**).



Fig. 30.82: Attitude in C₆ spine injury.

C₇ lesion causes abducted internally rotated arm (deltoid, teres major, subscapularis) with forearm flexed and pronated (biceps and brachioradialis) (**Fig. 30.83**).

C₈, T₁ lesion causes paralysis with *claw hand* like deformity.

Below T₁ up to T₁₀ level, chest, lower limbs are paralysed corresponding to the level of injury.



Fig. 30.83: Attitude in C₇ spine injury.

Below L₁ level, *cauda equina* is injured causing flaccid paralysis below knee level with loss of sensation around perineum, dorsum of legs with retention of urine.

In injury above T₂, breathing is *abdominal* as all intercostal muscles are paralysed.

Initially all reflexes will be lost due to spinal shock but gradually many of them reappear in 3 weeks depending on level of injury.

Sensations, muscle power, reflexes, rectal examination for fracture coccyx and sphincter tone, distended urinary bladder should be checked.

Spinal column should be examined carefully for swelling (haematoma), bruises, deformity.

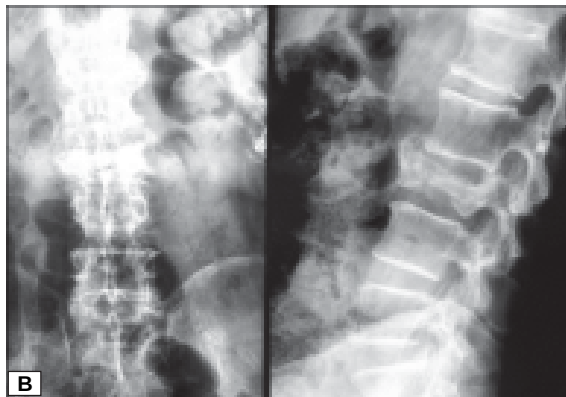
Finger is run along the spinous process—*Holdsworth test*. Tear in the interspinous ligament causes gap in the line of spine which suggests unstable fracture. In fracture dislocation spinous process will be prominent *below* the displaced vertebra. Vertebra *above* the injured one will be having prominent spine in compression fracture. Tenderness over spine should be checked using pressure.

Note: Abnormal mobility and movements *should not be checked* in spinal injuries. Other injuries (head, thorax, abdomen, pelvis, fracture limbs) should be assessed.

Stability of spine is maintained by *anterior component* (body of vertebra, intervertebral disc, anterior longitudinal ligament) and posterior ligament complex (supraspinous, interspinous ligaments and ligamentum flavum).

Fracture Spine

It can be **stable** without spinal cord injury or **unstable** fracture with or susceptible cord injury. Posterior ligament complex maintains the stability of the spine (supra, interspinous ligaments) (**Figs 30.84A and B**).



Figs 30.84A and B: X-rays showing spine fracture.

Burst fracture: It is stable vertical compression fracture.

Extension injury: It is stable fracture in atlas/axis (anterior ligament is torn)/lumbar region (fracture lamina).

Flexion injury: Here body of vertebra fractures with intact posterior ligaments causing stable fracture. It is common in lumbar vertebra but rare in neck.

Shearing force injury: Here there is rotational with flexion unstable injury causing fracture of vertebra and posterior facet.

Incomplete fractures: These are fractures of spinous processes, transverse processes and laminae due to direct injury. Spinous process fractures are common in thoracic spine; transverse process fractures are common in lumbar region.

Jefferson fracture: It is burst fracture of C₁ (atlas) without neurological deficit but vertebral artery may get involved causing *Wallenberg syndrome* (involvement of same side cranial nerves, Horner's syndrome, ataxia, loss of pain and temperature sensation on opposite side).

Hangman fracture (Wood Jones): It occurs in judicial hanging with distraction fracture of C₂ (axis).

Clay Shoveller's fracture (Australia): It is avulsion injury of spine of C₇ vertebra.

Chance/jack knife/seat belt injury: It is due to forced forward flexion causing transverse fracture of posterior element with compression fracture of vertebral body. If posterior injury causes failure of ligament complex it becomes unstable.

Whiplash injury: Neck is forcibly hyperextended and flexed due to rear end collision. It causes injury of soft tissues, ligaments and nerve roots. *Features:* Pain and stiffness in neck; limitation of neck movements; headache; hoarseness of voice due to recurrent laryngeal nerve injury; dysphagia; radiating pain in back and shoulder, paraesthesia.

Spinal Cord Injuries

Spinal shock due to concussion: It causes initial complete paralysis but later complete recovery.

Contusion of cord: It causes partial damage of the cord and features of same.

Root transaction: It is due to nerve root or cauda equine injury. Flaccid paralysis may recover.

Complete transaction of the spinal cord: It causes total loss of spinal cord function below the level.

Intraspinal haemorrhage: *Haematomyelia* is bleeding into the cord can cause paralysis. *Haematorrachis*

is bleeding in extramedullary region which causes initial spinal irritation later progressive spinal cord damage from below upwards.

Incomplete injury to spinal cord (partial trisection) causes various syndromes: (1) *Anterior cord syndrome*—There is complete motor loss, loss of pain, temperature below the level of injury; but intact deep touch, position and vibration sense. 2) *Central cord syndrome*—It is due to hyperextension injury in cervical spine involving central part of the spinal cord. It shows 50% gradual recovery. 3) *Brown Sequerd syndrome*—Here there is transaction of one half of the cord causing ipsilateral complete paralysis; hypoaesthesia of opposite side.

Spinal Diseases

Spina bifida occurs in newborn. Secondaries in spine occur in aged. Disc prolapse occurs in middle aged.

Disc prolapse is common in males; osteomalacia is common in females.

History of trauma, pain, mode of onset, nature and site of pain, aggravating and relieving factors, and radiation should be asked. Pain during lifting the weight occurs in disc prolapse. Pain may increase during coughing, sneezing, straining. Night pain is common in tuberculosis. Pain may radiate along the course of nerve. In tuberculosis of cervical spine, pain refers to occiput and arm; thoracic spine refers along intercostal nerves; thoracolumbar spine refers to girdle or epigastric region; lumbar spine to hip and leg. In ankylosing spondylitis pain occurs along the course of sciatic nerve alternatively one side and other.

Swelling: Meningocele, lipoma over spine, dimpling, tuft of hairs, cold abscess - duration, progression, pain should be asked.

Examination

Inspection

Attitude: Entire back should be examined from behind and from sides. Positions of head, shoulders, margins of body from axilla to iliac crests, curvatures of spine (forward bending—kyphosis; backward bending—lordosis; side bending—scoliosis) should be noted. In tuberculosis of cervical spine, child supports the

head with both hands under the chin; gibbus is seen in thoracolumbar tuberculosis. Prominent sacrum with deep transverse furrows between iliac crests and ribs is seen in spondylolisthesis. Gait should be checked.

Swelling should be assessed for all features like size, shape, and impulse on coughing. It may be cold abscess, spina bifida, meningocele, etc.

Features of paraplegia in tuberculosis of spine may be evident.

Palpation

It should be done in standing/sitting as well as in prone position.

Tenderness is elicited by running finger in midline from above downwards along the spine. It is also elicited by pressing the side of the spinous process to rotate the vertebra. Gentle blows on the side of the spine may elicit tenderness. *Anvil test:* Sudden jerk applied over the head of the patient causes pain/tenderness in the spinous process (this should be done cautiously). Tenderness can be also elicited by percussion over spine (**Figs 30.85 to 30.87**).

Swelling: Cold abscess may occur in neck, mediastinum, thoracic wall, psoas region; pelvis, etc. Meningocele may be present in spina bifida.

Rigidity in paraspinal region/paraspinal spasm should be checked by gentle running the thumb or index finger. Wasting of paraspinal muscles is also important.



Fig. 30.85: Gentle blows can be used to elicit tenderness.



Fig. 30.86: Pressing the lateral margin of the spine causes rotation and tenderness.



Fig. 30.87: Percussion also is used to elicit tenderness.

Movements

Flexion: It occurs in lumbar region up to the extent of obliteration of lumbar convexity. It is checked by asking the patient to lean forward with knees straight while examiner keeps his hands over the spine to note the spine movements. Children may be asked to pick up a coin from the ground.

Extension: It occurs in lumbar and thoracolumbar region. Patient is asked to lean backwards to look at ceiling. In children lumbar spine has got more mobility especially extension. Child is asked to lie down in the bed in prone position. Examiner places one hand over the dorsal spine to fix and with other hand lifts the legs together to elicit the range of extension.

Lateral flexion: It occurs with rotation of the vertebra in thoracic part. It is checked in standing position. Examiner holds the pelvis of the patient firmly to stabilise; patient is then asked to bend sideward to slide his hand with extended elbow along the thigh. In children: Child is asked to lie down in the bed in prone position. Examiner places one hand over the dorsal spine to fix and with other hand lifts the legs together and turns sideward to elicit lateral flexion.

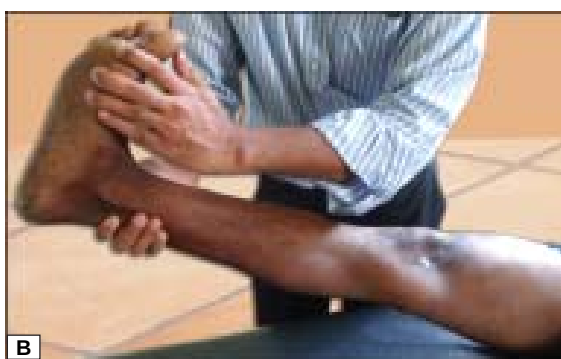
Rotation: It occurs in thoracic and cervical spines. Patient will sit on a chair to fix his pelvis and is asked to rotate his trunk right and left to demonstrate rotation. Nodding of head occurs at occipitoatlantic joint; rotation of head occurs at atlantoaxial joint (**Fig. 30.88**).



Fig. 30.88: Rotation movement of spine is checked in sitting position so as to fix the pelvis.

Straight Leg Raising Test (SLR)

Patient lying supine in the bed is asked to raise his leg with knee straight until he develops pain. If angle at which he develops pain is 40° , it is due to impingement of protruded intervertebral disc to nerve root. If pain develops beyond 40° , it is due to tension on the nerve root which may or may not be due to disc prolapse. There should not be compensatory lumbar lordosis to do this test. Both by active (by the patient) and passive (by the examiner holding at ankle) methods, it should be checked. **Lassegue's sign:** At the angle at which patient develops pain, ankle is dorsiflexed by the examiner passively which will aggravate the pain in case of disc prolapse or spine lesions but not in case of sacroiliac joint disease or sciatica (where SLR may be positive but not Lassegue's sign) (**Figs 30.89A and B**).



Figs 30.89A and B: Straight leg raising—SLR (active) and SLR Lassegue's sign eliciting (Passive).

Femoral Nerve Stretch Test

Patient in prone position, knee is flexed to cause the pain in front of thigh along the distribution of femoral

nerve which is due to protruded L_2 , L_3 disc causing stretching of femoral nerve root.

Naffziger's Test

Pressure on the jugular vein increases the pain of disc prolapse.

Lhermitte's Sign

Neck and hips are simultaneously flexed with the knees in extended position to cause sharp pain down the spine into extremities due to irritation of spinal duramater in case of cervical disc prolapse or extradural spinal tumour (**Fig. 30.90**).



Fig. 30.90: Lhermitte sign.

Aird Test for Malingering

Patient is asked to touch the toes in standing and if he is unable to do; then he is asked to touch toes in sitting position, which if he can means he is malingering.

Magnuson's Test for Malingering

Patient is asked pin point the site of pain and is marked. Patient's attention is diverted by throat, neck, and other examinations and once again is asked to show the site of pain. If he shows in different place he is malingering.

Complete neurological examination is a must—sensation, muscle power, reflexes, and tests for continence of urine.

Tests for Sacroiliac Joint

Tenderness in this joint test is elicited by placing the thumb over the dimple of the joint during bending. It is also elicited by compressing two iliac crests.

Forward bending and rotatory movements are painful in standing but not in sitting position.

Genslen's test: Hip and knee of diseased side is flexed and normal side hip is hyperextended on the edge of the bed to exert rotation of sacroiliac joint to elicit pain (**Fig. 30.91**).



Fig. 30.91: Genslen's test.

Gillie's test: While patient is in prone position, examiner places one hand over normal sacro iliac joint to steady the pelvis and using other hand diseased side thigh is passively hyperextended to elicit pain (**Fig. 30.92**).



Fig. 30.92: Gillie's test.

Pump handle test: Examiner places his hand over the testing side shoulder of the patient. Examiner with the other hand holds the leg below the knee and flexes knee and hip towards opposite shoulder across the abdomen which elicits severe pain on the diseased side (**Figs 30.93A and B**).

Lassegue's sign of SLR will be negative in sacroiliac joint disease.



Figs 30.93A and B: Pump handle test.

Investigations

- X-ray spine, X-ray chest.
- Blood tests: ESR, tumour markers.
- MRI is very useful.
- Myelography to see the disc prolapse or spinal diseases.
- Discography, epidurography are other investigations.

Ankylosing Spondylitis (Poker's Back, Marie Strumpell Arthritis)

It is a chronic, progressive disease of spine and sacroiliac joints, which is genetically predisposed as *HLA-B-27*. There is progressive restriction of movements of all joints in the spine. Patient cannot bend with

total stiffness and calcification of ligaments of the spine (*Bamboo spine*). Aortic valve disease, urethritis are other associations. Pain radiating to right and left lower limbs alternatively is typical. Patient stands with kyphosis with knees bent. Costovertebral ankylosis causes *poor chest expansion* (< 5 cms) leading to pulmonary complications. Eventually ankylosis of hip, knee, temporomandibular joints occur. *Condition is diagnosed* by clinical features, X-ray, ESR, positive HLA-B 27, negative rheumatoid factor.

Kyphosis

It means there is an exaggerated curvature of thoracic spine with obliteration of lumbar lordosis. There is excessive posterior convexity of thoracic spine.

Causes: Tuberculosis of spine—angular—*Gibbus type*; Adolescent kyphosis; post-traumatic (*Kummell's*), ankylosing spondylitis, osteoporosis, secondaries, Paget's disease, eosinophilic granuloma of vertebra (*Calve's disease*).

Types: (1) *Angular—knuckle*—where one spine is prominent due to collapse of one vertebra. (2) *Angular-gibbus*—one or two vertebrae are involved. (3) *Rounded*—many vertebrae are involved. *Postural kyphosis* is seen in girls with flat foot, obesity. *Compensatory kyphosis* is seen in lumbar lordosis, fixed flexion deformity of hip, CDH. *Scheuermann's disease* is osteochondritis of vertebral epiphysis in adolescents involving T₆ to T₁₀. Epiphyses contain small, translucent *Schmorl's nodes*. *Senile kyphosis* is due to old age degeneration of the disc.

Scoliosis

It is lateral curvature of the spine usually associated with rotational deformity. It is common in *thoracic spine*. It can be thoracolumbar or lumbar. It can be transient, mobile, non structural or can be structural. Condition may be associated with cardiac anomalies.

Transient scoliosis: (1) *Postural*—It is commonest non structural type seen in girls with mild convex curve towards left. It disappears while bending forward. (2) *Compensatory*—It is to compensate pelvic tilt in short leg or hip disease which disappears on sitting. (3) *Sciatic*—It is due to pain of intervertebral disc prolapse.

Structural scoliosis: Here body rotates towards convexity of the curve; spine towards concavity. It progresses till the cessation of growth. On X-ray, *Cobb's angle* between margins of vertebrae is assessed for severity. Completion index is assessed by fusion of apophysis with iliac bones (*Reisser's sign*). Main curvature is called as *primary curve* with compensatory secondary curvatures above and below. Rib hump appears on flexion of spine. Chest opposite to posterior convexity is more prominent. It can be—(1) *Idiopathic*—It begins in infancy, childhood, adolescents. It may resolve or progress. Progressive type leads into ugly type deformity with rib hump in thoracic spine. (2) *Congenital*—hemivertebrae, fused vertebrae, absence ribs, fused ribs. (3) *Paralytic*—poliomyelitis, cerebral palsy, muscle dystrophy. (4) von Recklinghausen disease of neurofibromatosis is associated with scoliosis (33%) (**Figs 30.94A and B**).



Figs 30.94A and B: Structural kyphoscoliosis.

Spondylolisthesis

It is defined as slipping forward of one vertebra over the next lower vertebra, usually seen in L₄–L₅ or L₅–S₁ junction. It can be congenital (75%); degenerative (20%); traumatic (5%). Parainterarticular of L₅ is defective with fibrous tissue component. It causes sudden severe pain with lumbar lordosis and step like depression over the sacrum. Short trunk, flat buttock, prominent sacrum with a depression above, transverse furrow encircling body between ribs and iliac crest are the features. X-ray oblique of spine shows decapitated *Scottish terrier* sign.

Intervertebral Disc Prolapse (IVDP)

It is herniation of the nucleus pulposus of the disc

through the nucleus fibrosus of the disc, commonly at postero-lateral direction in one or both sides. It is common in L₄-L₅ or L₅-S₁ region. Prolapse of L₄-L₅ disc compresses the lower nerve root-L₅. Prolapse of L₅-S₁ compresses the S₁ nerve root. X-ray shows findings only at late stage. Myelograms, MRI are diagnostic. It is the commonest cause of back pain. Always possible neurological deficits should be looked for. SLR test will be positive (**Figs 30.95 and 30.96**).

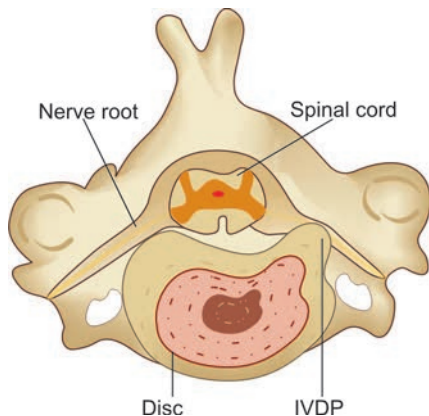


Fig. 30.95: Intervertebral disc prolapse posterolateral.



Fig. 30.96: MRI is the ideal investigation for IVDP.

Tuberculosis of Spine (Pott's Disease of Spine)

It is the tuberculosis of spine. It is the commonest type of joint tuberculosis (40%). It is common in children and adolescents; usually a secondary type. Commonest

area is *thoracolumbar region*—(T₁₀). Common site is *paradiscal part* of the vertebra. Occasionally central body, anterior surface, pedicle, lamina, spinous process may be involved. It is commonly haematogenous in origin.

In cervical vertebra: C_{6,7} is commonly involved. Neck pain, rigidity, brachial neuralgia, rust sign, tenderness and paraspinal spasm, restricted neck movements, retropharyngeal or posterior triangle cold abscess are the features.

In thoracic vertebra: Tuberculous metaphysitis is the commonest type. **Pathology:** Once intervertebral disc gets destructed, vertebral bodies collapse, causing *kyphosis* (forward bending of the spine). It is commonly angular type called as *gibbus*. If many vertebrae are involved then it is called as *rounded kyphosis*. There is destruction of bone, caseation, and *cold abscess* formation. Caseating fluid may trickle along the neurovascular bundle or along fascial planes leading to formation of cold abscess at different sites like psoas area (*psoas abscess*), paraspinal area. Destructed bone, granulation tissue, cold abscess may compress the spinal cord leading to *early onset paraplegia* (paraplegia in flexion). It can be reversed by treatment. Longitudinal gliosis, destruction of spinal cord will lead on to *late onset paraplegia*. It is irreversible type.

Clinical features: Typical military attitude with raised shoulder; Pain and tenderness in the spine; Paraspinal spasm confirmed by positive coin test; restricted spine movements; Deformity like gibbus; Cold abscess in different locations; Neurological deficits and paraplegia. *Paraplegia in flexion* is early feature with active disease and is due to compression or meningitis. *Paraplegia in extension* is late feature with a healed disease and is due to stretching of spinal cord, or longitudinal shrinkage of spinal cord due to gliosis (**Figs 30.97 to 30.99**).

Spina Bifida

It is failure of enfolding of nerve elements within the spinal canal during developmental period. It is usually seen in lumbosacral region. There is failure of fusion of the one or more posterior vertebral arches. It is often associated with other anomalies.

Sites: (1) Lumbosacral. (2) Thoracolumbar.



Fig. 30.97: Coin test in tuberculosis. Paraspinal spasm prevents proper spine functioning.



Figs 30.98A and B: Gibbus type of ankylosis in tuberculosis of thoracic spine.



Fig. 30.99: Tuberculosis of spine L₄, L₅ lateral view X-ray.

Types: *Spina bifida occulta*—There is dimpling of skin with dermoids, lipomas in the site. Impulse on coughing can be seen. Initially there is no neurological deficit but later tethering, traction on dura, infection can lead on to neurological deficits. *Membrana reunions*, a fibrous band between skin and spinal theca will be present. *Spina bifida aperta*—Here neurological deficit is present. It may be **myelomeningocele** wherein spinal cord and nerve roots are in the sac. It may be **meningocele** wherein sac consists of meninges and fluid only. Meningocele is brilliantly transilluminant. Myelomeningocele is not transilluminant. **Syringomyelocele** shows dilated central canal of spinal cord which lies within and is adherent to the sac. **Myelocele** has central canal of spinal cord opened into the skin discharging CSF. It is commonest and is not compatible with life. Paralysis, incontinence, sepsis soon sets in.

Clinical features: Motor paralysis; sensory paralysis; visceral paralysis with incontinence of urine and faeces; swelling in the spine at the site of the lesion may be lipoma or dermoid with impulse on coughing; bony defect at the site; later hydrocephalous (**Fig. 30.100**).



Fig. 30.100: Spina bifida.

Spinal Cord Tumours

Extradural—Here commonest is secondaries. Primary may be bronchus, breast, prostate or kidney.

Intradural: (1) *Extramedullary* (75%)—Neurofibroma is the commonest type (males) from posterior nerve roots. Spinal meningiomas are seen in females. (2) *Intramedullary*—Diffuse gliomas are commonest type. Ependymoma, vascular lesions are others. Common site is cervical region.

Clinical features: Paraplegia, neurological deficits, pain, urinary disturbances. Compression of spinal cord can occur. X-ray, myelography, MRI are investigations. CSF below the block may be yellow and proteinaceous—*Frolin's syndrome* (**Fig. 30.101**).



Fig. 30.101: MRI showing spinal cord tumour—ependymoma.

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